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The Sir Richard Stawell Oration.[1]

By C. H. KELLAWAY,
Melbourne.

IT is to an unhappy event that I owe the honour of delivering this, the fourth Sir Richard Stawell Oration, for we were to have had the pleasure of listening to Dr. Felix Meyer, whose death on August 31, 1937, deprived us of one of the most cultured and talented members of our profession. Dr. Meyer had chosen as his subject "A Consideration of Some Changing Aspects of Medicine", and it was his intention briefly to trace the history of medicine from the Golden Age of Greece, through the darkness of the Middle Ages to the Renaissance and so to the heart of his subject—the rapidly changing conditions of today.

Dr. Meyer had obtained from Mr. William Stawell some particulars about Sir Richard's forbears which form an interesting addition to the brief sketch of Sir Richard's life which Sir Charles Bickerton Blackburn gave in the first Stawell oration.

The Stawells were a County Cork family until Ireland became a free State under De Valera. Sir Richard's great-grandfather was George Stawell, J.P., of Ballyveniter and Ballylought, who in 1776 married Elizabeth, daughter of Jonas Stawell, of Kilbrittain Castle. Among their children was Jonas Stawell of Old Court in the same county, who married Anna Elizabeth, daughter of the Right Reverend William Foster, Lord Bishop of Clogher, on February 14, 1805. These were the parents of

Sir Richard's father, Sir William Foster Stawell, who came out to Australia in 1842 and became Attorney-General and later Chief Justice and Lieutenant-Governor of Victoria, and who died, while on leave at Naples, on March 12, 1889. Sir Richard's mother was Mary Frances Elizabeth, only daughter of William Pomeroy Green, R.N., of Cotters House, County of Louth, Ireland, and Woodlands, Broadmeadows, Victoria. Her father had been wounded in the Burmese War and invalided as first lieutenant in the navy. He never really recovered from the effects of his war service and decided to come out to Australia in search of health. In 1842 he chartered a 500-ton sailing ship, the *Sarah*, and brought out his wife and their family of six sons and a daughter, together with a number of family retainers, to Australia. He commuted his pension for a Crown grant of 640 acres at Broadmeadows and established his home there.

On the same vessel William Stawell, then a young man of twenty-seven, who was to play so large a part in the history of his adopted country, came out to Australia. The *Sarah* was not particularly well found—its registration as A1 at Lloyds was altered after it had been chartered, and it was in fact lost on its next voyage. It is interesting to reflect how fraught with fate was this adventurous migration. The history of Victoria might have been very different without the resolute young Attorney-General who saw the State through the difficult times of the gold rush and the crisis of the Eureka stockade and who later framed the *Constitution Act*.

In 1911 Sir Richard's mother, who was only twelve when her family came out to Australia, collected and published (for private circulation) her mother's letters and many of her late husband's, together with her recollections of the early days in their new country. This charming and intimate narrative gives the reader a deep insight into the character of Sir Richard's parents. It was natural that people of such quality should transmit to their children a goodly heritage.

Sir Richard was the sixth child of a family of five sons and five daughters, and early gave evidence of the qualities which were to make him a great physician. He was educated at Marlborough, but was rather delicate and came back to Melbourne, where he studied medicine, graduating with first class honours in 1888. My predecessors have described his subsequent career and have indicated the many directions in which he exercised his talents while he progressed to the preeminent position which he came to occupy in our profession. It remains to me only to add my personal tribute to his memory.

No one who was a medical student during the years in which Dr. Stawell taught in the out-patients' department and later in the wards of the Melbourne Hospital could fail to have a lively sense of gratitude that it had fallen to his lot to come under the influence of so inspiring a teacher. Stawell's enthusiasm, his intense interest in every case, his faculty of keen and accurate observation, the curiosity that made him discontented with anything less than an exact diagnosis, and the lucid manner in which he marshalled the facts and arrived at a conclusion—these made him a great clinical teacher. He strove to make his students think for themselves. With what enthusiasm were their observations and deductions received when they were right, and with what amazement at their almost unbelievable stupidity when, as was not infrequently the case, they were wrong! But his sympathetic insight into the frail humanity of patients and students alike ensured for him not only our admiration, but our affection.

During the period between 1923 and 1935, as director of the Hall Institute, it was my privilege to be more closely associated with Dr. Stawell at the Melbourne Hospital. He had then been a member of the committee of management and chairman of the house committee of the hospital for many years. He was deeply interested in the development of the Hall Institute, saw clearly the value of a close association between clinical and academic research in medicine, and used his great influence with the committee of management to secure institute beds in the hospital for special clinical studies, and the maintenance of effective pathological, bacteriological and biochemical routine services for the hospital. In 1928 he was for a time president of the hospital and chairman of the board of the institute, and it was a source of great regret to us when he resigned because he could not continue to give so much time from his busy consultant practice. Despite the heavy demands which were continually being made upon him, Dr. Stawell always found time to listen to the story of our difficulties and could be relied upon for wise advice and generous encouragement.

The enthusiasm which Stawell brought to the practice of medicine he carried with him also in his outdoor pursuits. Two pictures of him come to my mind. He had gone on a fishing expedition with two or three of his younger colleagues. On the first day their presentation of the dry fly to the reluctant trout had been somewhat lacking in effectiveness. Next morning early the neophytes were lined up in a paddock adjoining the hotel for a little instruction in the art of casting. Stawell used here the same methods which he had successfully applied in the teaching of clinical medicine—a cast less poor than usual received its meed of praise and the performer was spurred on to greater efforts, but our unsuccessful efforts evoked the same amazement with which he had been wont to receive our less happy essays in diagnosis.

Another expedition — the party was gathered round a large pool for the evening rise. The trout were feeding freely, but on something very small. Most of us, with more enthusiasm than intelligence, flogged the water for about half an hour without result, but Stawell had observed that the fish were feeding on a small spent spinner. He presented the

appropriate fly and was rewarded by the capture of a fish.

When fishermen are gathered together the conversation tends to be mainly of fish; but not so when Stawell was of the company. His talk was always stimulating and reflected his judicial and well-furnished mind. What delightful parties those were, and how Stawell charmed us and all the strangers whom we met at the places where we stayed!

Our affection for Stawell was strongly mingled with pride in him. A member of the legal profession in his father's life-time expressed what his colleagues and juniors thought of Sir William when he said "We are proud of Stawell". We had the same feeling about Sir Richard. On public occasions and at medical meetings he had usually something worth saying and said it very well. His speeches were carefully thought out and beautifully expressed, with flashes of wit to enliven their seriousness. His face and bearing made him a distinguished figure in any gathering. We were proud of him because of his high attainments, his culture and his wisdom, but most of all because of the man he was—kindly, generous, ever thoughtful of others, the embodiment of our ideal of what a great physician should be.

The subject upon which I shall address you—medical research in Australia—is appropriate to this occasion, for when Dr. Stawell qualified in 1888 he had intended to devote his life to research. However, the financial depression of 1893 deprived him of his private income, and since there was then no opportunity in Australia to obtain even a modest competence in research, he turned to the equally arduous but somewhat more lucrative practice of clinical medicine.

For the purpose of this dissertation I shall define medical research as the attempt to add verifiable data to existing knowledge concerning the normal structure and functions of the human body and its reactions to environment, and concerning the natural history of human diseases, their recognition, pathology and treatment, and the means to be adopted to diminish their incidence. It will be observed that this definition is wide enough in its scope to include researches in medicine and surgery and all their special branches, including dentistry, and in all the fundamental sciences upon which they are based. In another sense it is narrow, for it excludes all data which are incapable of rigid proof. These have a place in the art but not in the science of medicine.

There are over five thousand medical men and women practising in Australia who are engaged continuously in building up clinical experience which facilitates their diagnosis of disease and assures them of the most satisfactory measures for its treatment. Much of this experience, though vital to their success as physicians, surgeons or specialists, does not necessarily add one jot or tittle to scientific medicine. A great deal of it consists of impressions which, even if true, cannot be conclusively proved. Much such clinical wisdom must pass with the passing of the individual by whom it was acquired. The problems upon which the practising doctor is engaged are complex, not easily defined or limited, and the conditions often cannot be controlled so as to yield clear-cut and easily repeatable results. Nevertheless he may make definite contributions to medical science. He may recognize and describe the symptomatology of a new disease or may add to the knowledge of diseases already well known by careful analysis of his observations in a series of cases followed during a long period of practice; he may throw fresh light on the nature and cause of symptoms or initiate new methods of treatment. If these facts are borne in mind it will be obvious that I cannot, in the course of this brief address, hope to follow the development of clinical medicine in Australia nor to review the numerous contributions which have been made in this wide field of activity; but we may in passing recall with pride the researches of Joseph Bancroft on filaria, those of Davies Thomas on hydatid disease, and the contributions of Hamilton Russell to surgery.

In a young country there is little opportunity for the development of scientific research. The pioneers are fully occupied in the struggle for existence, and many years must elapse before the conditions of life afford to suitable persons the necessary leisure and detachment to set about the business of adding to the world's store of knowledge. A few gifted individuals may in their leisure hours make fresh discoveries, but until the conditions are created for setting aside persons with special gifts for whole-time research, advance must be slow and difficult. In these early stages men of ability must direct their energies to the adaptation of the knowledge of the old world to the special requirements of a new country and to the development of social life to the level at which organized advance becomes possible.

The early development of medical research in Australia was closely related to the formation and growth of the university medical schools, and owes a large debt to the men who made the creation of these schools their life work.

In 1883, when the Sydney medical school was founded, Thomas Peter Anderson Stuart, then twenty-seven years of age, came out to Sydney as Professor of Anatomy and Physiology. He had already done important original work, but now devoted his energies to the development of the medical school, of which he became dean, and of the closely associated Prince Alfred Hospital. He also found time to play a large part in the organization of health measures in the State of New South Wales.

In Melbourne the medical school had been founded twenty years earlier. George Britton Halford, who also had a distinguished record of research, was its first professor of anatomy, physiology and pathology. Its real development as a medical school dates from the appointment in 1886,

as dean, of Harry Brookes Allen, one of its own graduates, who in 1882 had become professor in anatomy and pathology. Allen, apart from an interlude from 1891 to 1896, when Halford was again dean, guided the destiny of this young school until two years before his death in 1926.

The Adelaide medical school was founded in 1884, and Dr. Archibald Watson, who made numerous contributions to surgical anatomy, was made Elder professor of anatomy. The creator of the school was Edward Charles Stirling, who was at first lecturer in and later professor of physiology. Sir Edward Stirling was a man of great versatility who distinguished himself in several fields of scientific endeavour. He was professor of physiology and had been made a Fellow of the Royal Society for his researches on the large extinct marsupial *Diprotodon australis*.

It was not to be expected that a large volume of research would at once emanate from these young medical schools. The primary object for which they had been created was that of training well-equipped medical practitioners, and the energies and time of the professors were naturally absorbed in teaching and organizing, but little being available for the search for new knowledge. Furthermore, in the early years the medical professors were compelled to take as their province not one, but two or three subjects. No money was available to provide either an adequate staff of lecturers and demonstrators or apparatus, materials and technical assistance for original work. Despite these disadvantages, we may well be proud of the early achievements of our medical schools in research. The investigation not only of specifically Australian problems but also of those of more general importance was made possible by the devotion of professors and lecturers who inspired their students and demonstrators to work with them, often during vacations.

Australia was exceedingly fortunate in the men who came from overseas to work in these young medical schools. Let us take an example from the early history of the Sydney school. Anderson Stuart had as successive demonstrators or lecturers in physiology three young men who subsequently attained great eminence. All were knighted for their services to medicine, and two became Fellows of the Royal Society. Alexander MacCormick was to develop rare clinical wisdom and amazing operative dexterity and to become the foremost surgeon in Australia. Almroth Wright became professor of pathology at the army school, Netley, and afterwards principal of the institute of pathology at Saint Mary's Hospital, London, where he gathered round him a brilliant group of young workers. He made numerous contributions to knowledge, notably the development of antityphoid vaccination, which has saved countless lives, and studies of the opsonins—"feast preparers", which encourage the phagocytes of the body in their task of devouring invading organisms.

The name of Charles James Martin occupies a special position in the history of medical research in Australia. After being lecturer in physiology in the University of Sydney he became, on the resignation of Professor Halford in 1897, lecturer in physiology at Melbourne. In 1901 he was made professor of physiology, but afterwards went to India to conduct investigations for the British Government on antityphoid inoculation and on plague. In 1906 he became director of the Lister Institute of Preventive Medicine. During the Great War he once more had close association with Australia, acting as chief of the pathological services of the Australian Imperial Force. Finally, after his resignation from the directorship of the Lister Institute, he returned to Australia to direct, for some two or three years, the Division of Animal Nutrition of the Council for Scientific and Industrial Research.

Martin had gifts which made him specially capable of carrying out first-rate research under adverse conditions, and no shortage of time, money, equipment or technical help could inhibit him. His mechanical ingenuity and resource surmounted all difficulties, and his wide interests and equally wide knowledge, his strong critical faculty, and the fact that he was always eager to learn from anyone, however humble, who had any special knowledge, made him an ideal worker in a young country. His enthusiasm was infectious, and both in Sydney and Melbourne he found disciples and co-workers.

In Sydney, McGarvie Smith was associated with him in his classical studies on Australian snake venoms, and in Melbourne, with Thomas Cherry, he did his important work on the separation of toxin and antitoxin by ultrafiltration. E. H. Embley learnt from him the technique of experimental medicine, and in the intervals of a busy practice made his well-known experiments on chloroform anæsthesia. Among his Melbourne students was Gordon Clunes Mathison, who did brilliant original work in Professor Starling's laboratory at University College in London, and who, but for his death in the War, would have played an important part in advancing medical research in Australia.

During this early period Sydney was fortunate to secure, in 1887, as its first lecturer in anatomy, James Thomas Wilson, who became professor in 1890 and occupied this chair for thirty years. He was made a Fellow of the Royal Society for his researches in anatomy and embryology, and left Sydney to become professor of anatomy at Cambridge. Wilson was an inspiring teacher, and among his distinguished pupils were Sir Grafton Elliot Smith, F.R.S., and John Hunter, who succeeded him in the Sydney chair.

Having made a fortunate beginning, the Australian medical schools have continued in the same tradition despite an increasing burden of teaching upon the professorial staff. It would be quite impossible for me to review the many and varied researches which have been carried out during the last thirty years, even in a single medical school.

For instance, at Melbourne alone I should have to describe physiological researches on a variety of subjects by Professor W. A. Osborne and his pupils; the biochemical researches of Rothera and of Professor W. J. Young and his pupils, including some excellent dental research; the numerous pathological studies which have come from Professor MacCallum's department, notably the work of E. S. J. King, who has twice won the Jacksonian Prize of the Royal College of Surgeons; the work of the bacteriological department under Dr. R. J. Bull and Professor Woodruff; and finally the brilliant morphological and anthropological studies of Professor Wood Jones.

The development of state medicine has also played a significant part in the advancement of medical research in Australia. In its early period the organization of public health measures and the application of the principles of preventive medicine to local problems fully occupied the time of the medical officers of health, but later much important work was done. In New South Wales, for example, John Ashburton Thompson, who became chief medical officer in 1896, made important contributions to epidemiology, notably his work on the history of leprosy in Australia (1894 and 1897). A little earlier the State Board of Health Laboratories, which later became known as the Bureau of Microbiology, were established. From this institution much valuable work has originated, for example Tidswell's epidemiological studies on plague (1901-1906), which pointed to its transmission by the rat flea; the work of Cleland, Campbell and Bradley (1917) on "X" disease—an obscure virus infection of the central nervous system which has so far been met with only in Australia; and the studies of Eustace W. Ferguson on various insect carriers of disease.

Federation in 1900 saw the beginning of further advances. The Federal Department of Health took charge of the quarantine services and furthered the statistical investigation of disease. The present Director-General of Health, Dr. J. H. L. Cumpston, made important contributions to epidemiology, the best known of which are his "History of Smallpox in Australia" and his studies on the Australian incidence of numerous other infectious diseases.

The laboratories division of the Commonwealth health services was instituted in 1915 and a central laboratory situated in Melbourne was shortly built for the manufacture of biological products. In the early development of this laboratory Dr. W. J. Penfold, who later became director of the Baker Institute of Medical Research, played an important part.

The conception of a State laboratory must be attributed in the first place to Sir Harry Allen. When Allen returned from Europe in 1891, in a report presented to the State Parliament he recommended "that the Government be invited to establish an Institute of Preventive Medicine . . . This should be created as a national necessity and placed in special departmental correspondence with the Board of Public Health." The sort of institute which Allen envisaged was a miniature Pasteur Institute. Some years later Dr. Cumpston read this report, was seized with the importance of this recommendation, and directed his energies to the establishment of a central laboratory. The serum laboratories, which started in a small way on the top floor of the Hall Institute building, are now a great laboratory at Parkville, which has undergone further development under the present director, Dr. Grantley Morgan. Apart altogether from its commercial activities and the large amount of early work which was done on industrial serological problems, it has recently been possible to carry out researches of more general interest. A further forward step has been taken in setting aside a number of workers for whole-time research. The first of these, Dr. E. V. Keogh, was appointed some years ago and was seconded to Dr. Burnet's department at the Hall Institute for some two and a half years, during which time he worked on virus problems, notably on vaccinia, on the production of immunity to virus diseases, and on a filtrable virus causing a sarcoma in birds, first described by Peyton Rous. The interest in this last study lies in the fact that it provides evidence of the essential unity of types of newgrowth, whether they affect epithelial cells or connective tissue, for Keogh has shown that this virus can grow in epithelial cells and cause typically cancerous lesions.

Although these laboratories are developing a strong research side, it is their present policy to aid and cooperate in research carried out at other institutions, rather than to establish a large national research institute. We may illustrate this point by reference to the proposal which will shortly come into effect, of a research on immunity to the virus of poliomyelitis, which the Commonwealth Serum Laboratories will carry out in cooperation with Dr. Burnet and his colleagues.

In addition to the central laboratory, smaller diagnostic laboratories were set up in various centres in Australia. There are now ten of these, five of which are situated in the tropical regions of Australia, at Broome, Darwin, Cairns, Townsville and Rockhampton. These not only provide diagnostic services for the surrounding districts, but are also available as centres of research. This has recently been illustrated by the studies on leptospirosis in Queensland which followed the discovery of leptospiræ, by Sawers and by Cotter, in cases at Ingham, already recognized clinically by Morrissey and Leckie. A large number of workers of the Commonwealth and of the Queensland State service have been engaged upon this problem. It is obvious that the story is a very complicated one. Lumley (1937), from the Townsville Commonwealth laboratory, has shown that there were two distinct strains of leptospiræ in the Ingham outbreak, different from the classical strain of Weil's disease; and Derrick, in the Queensland State laboratories,

in cases investigated clinically by Clayton, of Pomona, has found leptospirosis of a milder type corresponding to "seven-day fever".

In 1909 an Institute of Tropical Medicine was established at Townsville under the direction of Anton Breinl. Two years later, when the staff had been increased and the institute had been given more adequate financial support, it was possible to take up the problem of the white man in the tropics, and interesting studies on climatic physiology were made by W. J. Young, Henry Priestley and others. This institute, after various vicissitudes, was replaced in 1932 by the School of Public Health and Tropical Medicine at the University of Sydney, under the direction of Dr. Harvey Sutton.

The Commonwealth Department of Health has concerned itself also with a number of other activities. It has subsidized and encouraged laboratory research at a number of institutions in Australia and has aided the anti-cancer campaigns which have been initiated in every State. It purchased ten grammes of radium, which has been available for investigation and treatment and has helped to coordinate the activities in the various States and in the Dominion of New Zealand by the holding of an annual conference, where various aspects of the cancer problem are discussed.

The growth of hospitals, and particularly of those which function as clinical schools, is playing an increasing part in the advancement of medical science. In the large cities of the Commonwealth great hospitals have grown up, equipped with good pathological and diagnostic facilities, which offer excellent opportunities for clinical research. Naturally, the ideal conditions are provided by close geographic relationship between a hospital and a medical school—a condition of affairs which exists only in Sydney, where the Rockefeller Foundation has built a magnificent medical school in immediate proximity to the Royal Prince Alfred Hospital, and the Bosch Foundation has endowed whole-time chairs in medicine and surgery as well as in embryology and in bacteriology.

Failing such an association, another development is taking place which has already led to much important research. In 1916 the Hall Institute of Research in Pathology and Medicine was founded at the Melbourne Hospital, now the Royal Melbourne Hospital, and ten years later the Baker Institute was similarly founded at the Alfred Hospital, Melbourne. More recently the Kanematsu Institute was established at the Sydney Hospital. The Kolling Institute has since been founded at the Royal North Shore Hospital, Sydney, and only last year an institute of research in close relationship both to the university medical school and to the Adelaide Hospital has been established in Adelaide.

In these institutions research is not confined to clinical problems, and in most of them an attempt is being made to build up a staff of workers expert in different fields, whose whole time is devoted to research. In addition, young graduates are given the opportunity of doing part-time work on a variety of problems. There are great advantages inherent in this development, because it provides for a few men of ability the opportunity for whole-time research freed from the burdens of teaching, administration or practice, so that they may within the span of a few years put out a large volume of original work. It also affords opportunity for the development of clinical science and for the attack on clinical problems, with the resources of a well-equipped institution in immediate relationship to the hospital, and with people well trained in methods of research to advise and help.

Naturally much of the research which has been carried out at these institutes has been on Australian problems, for instance, the work of Hamilton Fairley, Harold Dew and Keith Fairley on hydatid disease, and that of Hamilton Fairley on the biting mechanism of Australian snakes; but other work has been of more general interest, for example the researches of Burnet on bacteriophage and on staphylococci, and those of Inglis, of Willis and of Cox on newgrowths. Important work has also emerged from the pathological departments of large hospitals, for instance that of A. H. Tebbutt at the Royal Prince Alfred Hospital, Sydney, and of Lionel Bull at the Adelaide Hospital.

Dr. Cumpston, in his inaugural address to the National Health and Medical Research Council in February of this year, has enumerated the various researches which are at present in progress in Australia. It is clearly impossible for me to review even briefly more than one or two of these, and I may be forgiven if I present to you on this occasion two fruitful lines of inquiry which have been in progress for some time at the Hall Institute. Naturally I am more familiar with these results than with work in other laboratories.

The study of virus disease is engaging the attention of workers in many parts of the world and is at present one of the most important "growing points" of knowledge. In this field Australia has made significant contributions through the work of F. M. Burnet, E. V. Keogh and Miss Lush. Viruses, which cause economically important diseases in plants and animals as well as in man, are extremely minute organisms which are capable of multiplying only within living cells. They are strict intracellular parasites. They vary greatly in size, but are small enough to pass through the pores of filters. The smallest of them are invisible to the highest powers of the microscope, but the largest, though still much smaller than bacteria, reach dimensions at which they are microscopically visible. The investigation of virus diseases is attended at the outset by the difficulty that these organisms will not multiply upon artficial media. It is therefore essential, in the case of those which cause human diseases, to find some animal in whose cells they will grow and in which they cause symptoms and recognizable changes.

ILLUSTRATIONS TO THE ARTICLE BY Dr. H. BOYD PENFOLD.

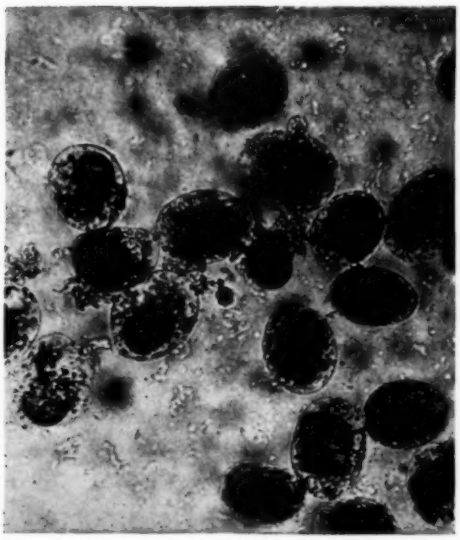

Figure I.
Tænia echinococcus ova, complete with external shell. (×180.)

Figure II.
Portion of the lower half of the small intestine of an artificially infested dog, showing hundreds of *Tæniæ echinococci* attached to the mucosa. (Natural size.)

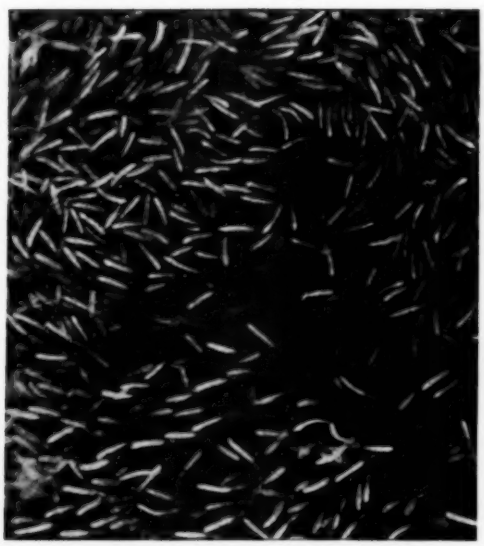

Figure III.
Numerous complete *Tæniæ echinococci*. (×2.)

Figure IV.
Complete *Tænia echinococcus*. (×15.)

ILLUSTRATIONS TO THE ARTICLE BY DR. PHYLLIS M. ANDERSON.

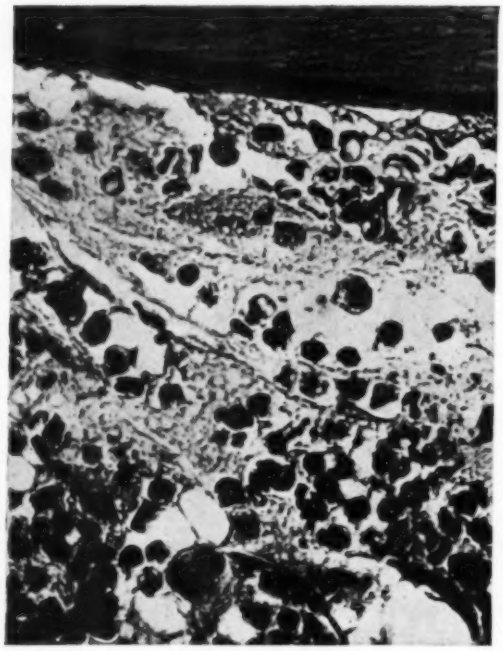

FIGURE IV.
Sternal marrow: erythroblasts, granular cells and one megakaryocyte.

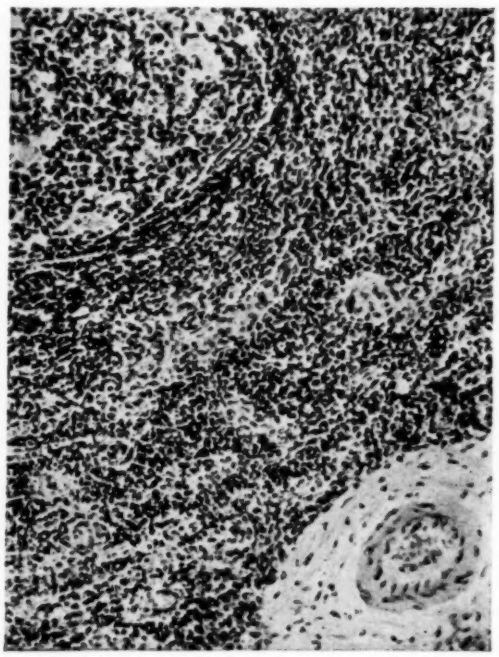

FIGURE V.
Spleen showing Malpighian body, with large pale cells and surrounding lymphocytes. Pulp cells with active phagocytosis. Vessel with wide adventitia.

ILLUSTRATION TO THE ARTICLE BY DR. S. L. SEYMOUR.

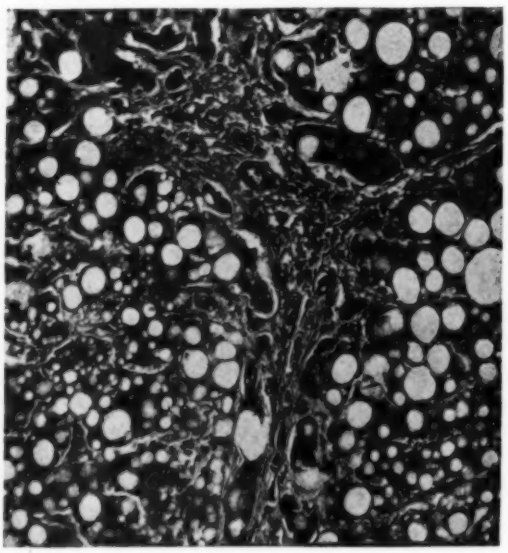

FIGURE I.

ILLUSTRATION TO THE ARTICLE BY DR. H. S. NEWLAND.

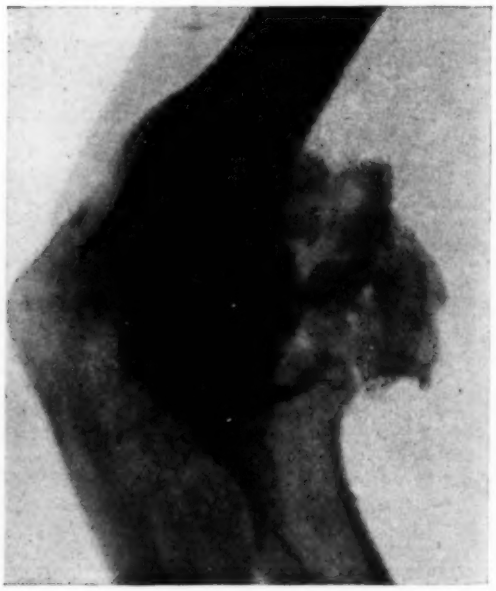

FIGURE I.

ILLUSTRATION TO THE ARTICLE BY DR. H. STUBBE.
("Recurrent Passive Collapse of the Lung.")

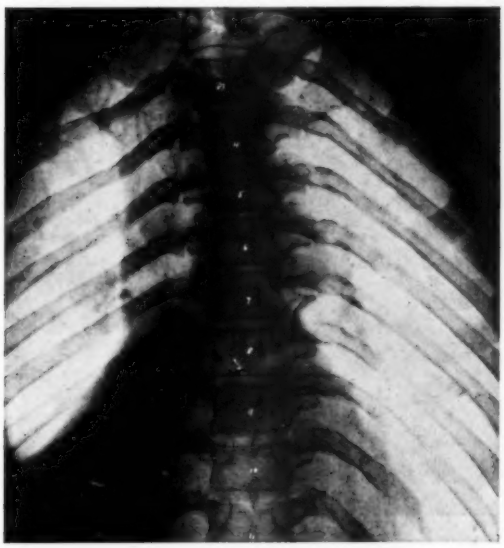

FIGURE I.

ILLUSTRATIONS TO THE ARTICLE BY DR. H. STUBBE.
("Elusive Calculi.")

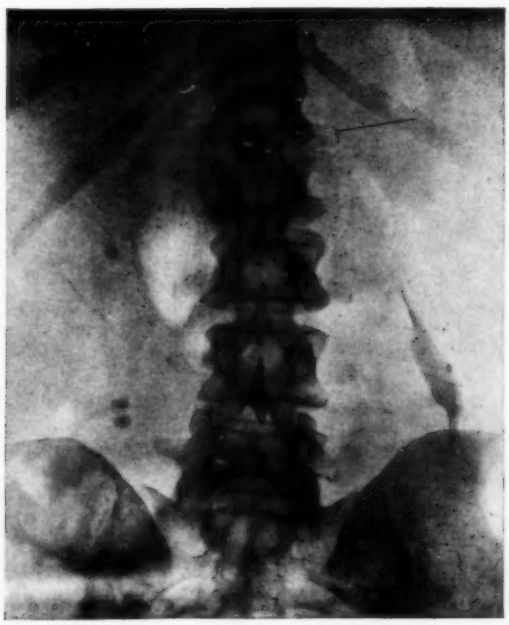

FIGURE I.

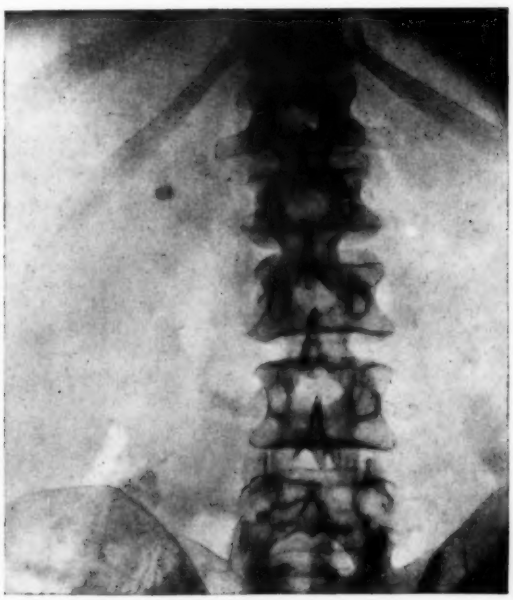

FIGURE II.

ILLUSTRATIONS TO THE ARTICLE BY DR. G. F. ELLIOTT AND DR. B. T. SHALLARD.

FIGURE III.

Plain skiagram. Note the density of the shadow and the calcification.

FIGURE IV.

Pyelogram showing both tumour and the distortion and displacement of the calyces.

ILLUSTRATION TO THE ARTICLE BY DR. JOHN McGEORGE, DR. GILBERT PHILLIPS AND DR. OLIVER LATHAM.

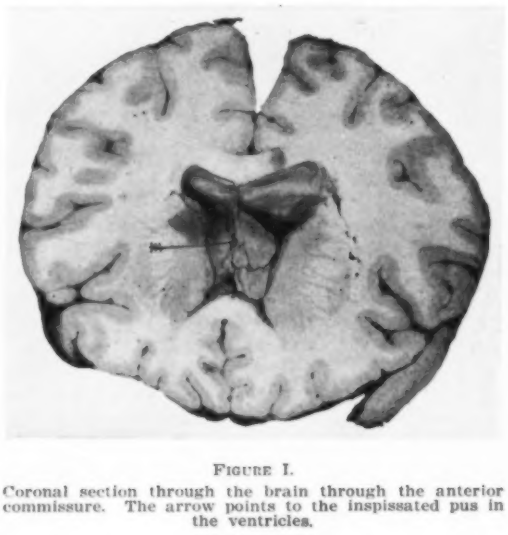

FIGURE I.

Coronal section through the brain through the anterior commissure. The arrow points to the inspissated pus in the ventricles.

ILLUSTRATION TO THE ARTICLE BY DR. H. FLECKER.

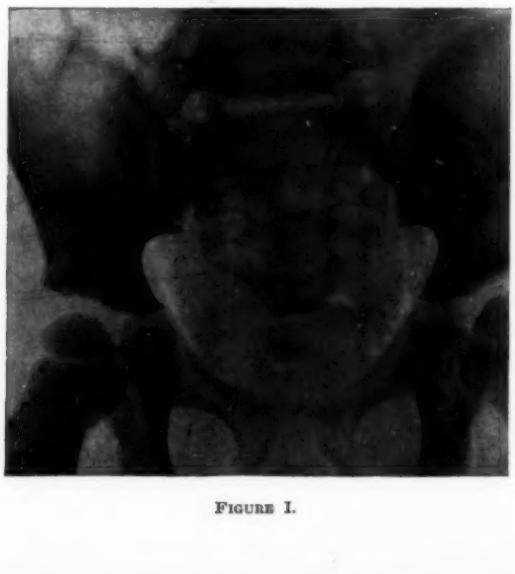

FIGURE I.

When, four years ago, Dr. Burnet was in England working at the National Institute for Medical Research, he utilized the discovery of Goodpasture, that some viruses would grow on the chorio-allantoic membrane of the developing hen egg, and devised an ingenious method, which has made possible much of the work that has since been done in his laboratory. The developing egg is lined by two membranes—a membrane in contact with the shell and a richly vascular inner membrane, the chorio-allantois, which is the breathing organ of the developing chick. The egg contains a large air space at one end, and Burnet discovered that it was possible to detach the chorio-allantoic membrane from the shell membrane by the creation of a large new artificial air space. In this way there is provided a large flat area of living tissue which is easily accessible for inoculation and upon which many viruses can be grown. This new artificial air space can be made, the membrane inoculated, the shell sealed up and the incubation of the egg continued for two to four days without the development of the chick being arrested. At the end of this time the egg is opened and the changes in the membrane are studied. The value of this method lies in the fact that when viruses are inoculated upon this surface they enter the cells and multiply there, causing proliferation and damage and thus producing small lesions on the surface of the membrane which can be seen with the naked eye and which are different in appearance and structure for different viruses.

In many ways the viruses behave like the larger bacteria which cause human diseases. The defence reaction of the body against them is similar to that against bacteria. Antibody is produced, and when it is present in sufficient amount in the blood and tissues an animal is thereby rendered immune to the attacks of the organism, whether it be a virus or a bacterium which has caused the immunological response. Now Dr. Burnet found that a mixture of virus with the corresponding antiserum when inoculated onto the egg membrane produced a much smaller number of lesions than was produced by the virus alone. It is thus possible to find how much antibody is present in the blood of an animal or man.

Some viruses do not cause changes on the egg membrane, but gain access to the circulation of the developing chick and cause its death, at the same time producing characteristic lesions in its tissues. In these cases also the developing chick can be used both for the propagation of the virus and for the titration of sera for antibody content.

The method has two other uses: it offers a means of obtaining quantities of bacteria-free virus and could be used to procure quickly large stocks of virus for vaccination against smallpox. It also provides a useful diagnostic method. Dr. Burnet was able, for example, to isolate and identify in this way the virus of acute laryngo-tracheitis, an economically important disease of poultry, from an outbreak which occurred recently in New South Wales. The results of this inquiry, which was undertaken at the request of and in cooperation with the State Veterinary Department of New South Wales, yielded interesting information about the epidemiology of the disease and suggested a sound method for its control.

The two main human virus diseases upon which work has been carried out in Dr. Burnet's laboratory are psittacosis and influenza. The first disease has caused serious outbreaks in America and Europe, though so far only one small outbreak has been recognized in Australia. Until the studies of Meyer in California were carried out it was thought to be an occasional disease of parrots accidentally transmissible to man. Meyer showed that the American outbreaks arose from enzootic disease in Californian aviaries, where most of the budgerigars distributed through the United States were bred. He found that a surprisingly large proportion of aviaries were infected, even among those in which the birds appeared to be healthy. Burnet has carried the story a step further by showing that infection in Australia among wild parrots and cockatoos is almost universal. The study of a small human epidemic by Burnet, in collaboration with Dame Jean Connor, has thrown further light on the natural history of the disease. The virus infects most young birds in the nest, but under normal conditions they survive the infection, though they may carry the virus for long periods. If captured nestlings are placed under bad conditions the disease may develop in a form severe enough to be fatal, and these birds are a real source of danger to man. The facts which have been obtained are sufficient to define the public health measures necessary to prevent the occurrence of outbreaks of the human disease.

The history of investigations concerning the nature of influenza goes back a good many years. In the great epidemic of 1890 the bacillus of Pfeiffer was isolated and was believed to be the cause. In the pandemic of 1918 many bacteriologists thought that a virus was responsible, but it was not till 1933 that Laidlaw and his colleagues at Hampstead showed that the disease was caused by a filtrable virus which could be transmitted to ferrets, and thus had a method at their disposal for further investigation. The primary importance of this work was to define the disease clinically. Previously, any acute but transient febrile disease of the respiratory tract was liable to be labelled as influenza. The virus is responsible for acute and widespread epidemics only, and not for sporadic disease. During such epidemics most people get infected either clinically or subclinically. They become temporarily immune and the epidemic rapidly dies down.

Since Burnet was able to grow the virus upon the membrane of the developing hen egg (a discovery which was made almost simultaneously at Hampstead) a great deal of further information about this important respiratory disease has been accumulated. Perhaps the most important point

that has been ascertained is that different epidemics are caused by different strains of the virus. For instance, the strain isolated from the Melbourne epidemic of 1935 differs from that isolated in London in 1933, and it is possible that the strain which caused the pandemic of 1918 was a third type.

When the virus is grown upon the egg membrane for many passages it changes in character—loses its virulence for the ferret, for mice and for man, but still retains the power of causing the production of antibody. In the attempt to produce artificial immunity this egg-adapted virus has been injected intranasally in human beings without causing any symptoms; but since there has been no epidemic since 1935 we cannot tell whether this procedure will be effective in protecting the population during an epidemic. If the method is to be successful it is probable that several strains of egg-adapted virus will have to be used, since there is no guarantee that the next epidemic here will be caused by a virus antigenically similar to that of 1935.

The second series of researches is the outcome of experiments upon the venoms of Australian snakes. We had recently observed that the effects of the venom of the Australian copperhead upon the circulation resembled closely those caused in poisoning by histamine. In 1935 Dr. Wilhelm Feldberg, who had already carried out fruitful work on the relation between anaphylactic shock and histamine poisoning, joined in the inquiry, and we were able to show that many of the symptoms of copperhead poisoning are to be explained not by the direct action of the venom itself, but by the liberation of histamine from cells injured by the venom.

Histamine is a normal constituent of many tissue cells. It is present in them in an inactive form, but if the cell envelope becomes permeable as the result of injury it can be set free into the tissue spaces or into the blood stream and may give rise to profound visceral and circulatory changes. These statements rest upon two lines of evidence. We were able to show that organs perfused for many hours with physiological saline solutions *in vitro* gave up none of their histamine, but that if venom was injected into the perfused organ or if the organ had been taken from an animal poisoned with venom, a large proportion of its histamine was set free and appeared in the perfusate. The second line of evidence was obtained by further analysis of the action of the venom in several species of laboratory animals. We found that the differences in symptomatology in these depended in part upon the variation in sensitivity of different tissues in these animals, both to the action of venom in causing histamine to be set free and to the action of histamine itself. In the guinea-pig, for example, changes in the lungs, and in the cat changes in the lung vessels, play an important part in causing the symptoms both of venom and histamine poisoning, and large amounts of histamine may be liberated by snake venom from these perfused organs. In the dog, characteristic changes in the liver are an important feature in poisoning by histamine and by snake venom, and perfusion experiments showed that from this organ also a large proportion of its histamine was liberated by venom.

The action of snake venom is not, however, confined to these special organs, for histamine is liberated from many tissues in the body and exerts its action at the site of liberation. For example, poisoning by snake venoms is attended by symptoms resembling those of circulatory shock; and there is a loss of fluid from the vessels and a diminished amount of blood in effective circulation. This loss of fluid is indicated by concentration of the corpuscular elements in the blood and is a well-known feature of histamine shock. We have been able to show that the circulatory shock of snake venom poisoning is associated with the same changes in the blood.

The similarity between the symptoms caused by snake venoms, bee venom, by a bacterial toxin and by protein split products is to be explained by the fact that they act by injuring the cell and liberating histamine, since it was shown in our laboratory by perfusion experiments that these various substances all release histamine from the tissues.

So far we know only of one pharmacologically active substance produced by injury—histamine. Its liberation is regarded as the first line of defence against injury. This view was put forward by Sir Thomas Lewis to explain the reaction of the vessels in the human skin to a variety of injurious agents, and he was able, by simple but ingenious experiments, to produce a large amount of indirect evidence in its support. The experiments at present in progress are providing direct evidence of the truth of this conception.

In discussing the progress of scientific medicine in Australia we cannot neglect mention of the rapid progress made in this country by veterinary science. Veterinary science has advantages over medicine in that the subject of disease may be used for experiments not desirable in man, and that when an infection is being investigated it is usually possible to reproduce the disease experimentally in animals of the same species. The parallel advance of veterinary science is of great advantage to the progress of medical science and has stimulated the study of infective diseases which are common to animals and man. Among a number of recent Australian discoveries in the veterinary field there are, for example, the studies by Turner of the minute organism which causes contagious pleuro-pneumonia of cattle and his demonstration of what the late Professor Gilruth once termed the unholy alliance between an animal (the liver fluke) and a plant (an anaerobic bacterium) in the production of black disease in sheep. The recent studies concerning the effectiveness of cobalt in curing coast sickness of cattle points to the significance of yet another metallic ion in physiology. This metal does not appear to be essential to all animals, since rabbits, which run on the areas where disease in sheep and cattle is prevalent, do not suffer any deficiency. There is no evidence of any similar deficiency

disease in man, but it is interesting to recall the rôle of copper in the treatment of anæmia.

When the volume of original work in a country becomes large, local publication must necessarily follow. Some of the early work was published in the proceedings of the various Australian Royal Societies, and much that is of local or clinical interest has been published in THE MEDICAL JOURNAL OF AUSTRALIA and its predecessors. A large amount of research is unsuitable for journals intended primarily for doctors in practice, and in order to be readily available to workers in other countries is best published in specialized periodicals which accept papers on one field of work only. For this reason much of the best Australian work is to be found in scientific journals published abroad. There are advantages and disadvantages in sending papers overseas for publication. On the one hand they are accessible to more people and are subjected before publication to valuable criticism not always obtainable in the country of origin; but, on the other hand, publication overseas, if proofs are to be submitted, rarely takes less than nine to twelve months, whereas with local journals two to four months between completion of the work and its publication is a normal time. The time of publication becomes important when groups of workers in different parts of the world are engaged upon the same problems.

The Commonwealth Department of Health has published a number of monographs and a monthly journal, *Health*. Important communications are also to be found in reports of the State departments of health. Some researches have been published in book form, and the Baker Institute has also put out several valuable contributions as monographs.

In 1924 there was founded, in the University of Adelaide, *The Australian Journal of Experimental Biology and Medical Science*. This, the first Australian journal wholly devoted to experimental biological science, we owe to the enthusiasm and creative vigour of Thorburn Brailsford Robertson, who was professor of physiology in the University of Adelaide from 1919 till his untimely death in January, 1930. This journal, which is to be found in nearly all the important medical libraries in the world, is steadily gaining in reputation and now provides for the publication of many papers which in earlier days would have been published abroad.

No account of medical research in Australia would be complete without reference to the work of outside bodies. Both the Rockefeller Foundation and the Carnegie Corporation of New York have contributed greatly to the progress of medical research in Australia. Between the years 1916 and 1923 the International Health Board of the Rockefeller Foundation carried out a hookworm campaign in Australia, Papua and New Guinea in cooperation with the Commonwealth Government and with the States where the investigations were made. The Rockefeller Foundation, in addition to building a medical school at Sydney, has during the last four years combined with the Health Department of the Commonwealth in subsidizing research on virus diseases carried out by Dr. Burnet and his colleagues.

The Carnegie Corporation of New York, which has helped in the progress of education in Australia, has recently made possible the establishment in Australia of a number of expatriated German scientists, two of whom, Dr. Feldberg and Dr. Lemberg, are engaged in medical research, the former at the Hall Institute, and the latter at the Kolling Institute at the Royal North Shore Hospital, Sydney.

The present year saw the development of what will probably prove to be the most important single forward step in the history of medical research in Australia. The Commonwealth Government, encouraged by the success of the Council for Scientific and Industrial Research in the prosecution of general research of national importance, decided to embark upon a new venture. In the *Gazette* of September 24, 1936, there was published by an Order in Council the constitution of the National Health and Medical Research Council. This was to take the place of the Federal Health Council, which since 1926 had been concerned with public health administration. The new council has as chairman Dr. J. H. L. Cumpston, the Director-General of Health of the Commonwealth. There are two other representatives of the Commonwealth Health Department, one representative each of the health departments of the States, one representative of the British Medical Association, one of the Royal Australasian College of Surgeons, one of the Association of Physicians, one of the Australian universities having medical schools, and two lay representatives appointed by the Commonwealth Government. In this body there are to be combined the functions of coordinating official preventive medicine, as represented by the health departments of the Commonwealth and States, and of coordinating and subsidizing medical research, both clinical and experimental, in existing institutions in Australia.

At its first meeting at Hobart (February 1 to 3, 1937) the council formulated its research policy. It was proposed that an annual subsidy of £30,000 should be provided by the Commonwealth Government, which should give the council the right to accumulate unexpended balances for use in later years. Money was not to be used for the erection of buildings, but in subsidizing specific researches at institutions approved and registered as such by the council, and particularly in encouraging young medical graduates to take up research as a career. This latter object was to be achieved by the establishment of junior fellowships in laboratory and in clinical research with a tenure of not more than three years at a salary of £500 *per annum*. These fellows would work under direction at an approved institution, and a few of the best of them might look forward to permanent appointments as senior

research fellows at a salary of not less than £1,000 a year. Young workers of exceptional ability might be sent abroad as travelling fellows to acquire experience in other countries.

Grants normally of the value of £250 a year would be made to medical graduates for part-time clinical research. Grants could also be made for various other purposes which would facilitate research—to individual workers for technical assistance, to institutions to supplement existing research endowments which were inadequate for their purpose, to Commonwealth or State health departments for occasional assistance in inquiries or for the prosecution of special inquiries, and to university departments to provide technical or other help and set free for research the professor or his assistants.

In general, research in any field of medicine, including dental science, would be recognized, but preference would be given to problems of special interest in Australia. To facilitate clinical research the collection of accurate and complete clinical records in hospitals was to be encouraged. The necessity of improving the level of technical assistance was recognized, and as a first step a register of technicians was to be compiled.

Applications for grants were to be scrutinized by a subcommittee consisting of the chairman, the representatives of the British Medical Association, the Royal Australasian College of Surgeons, the Association of Physicians and the universities, together with those members of the council in the State where the subcommittee held its meeting.

At the second meeting of the council further steps were taken for the coordination of research by the establishment of research committees on tuberculosis, on obstetrics and on tropical medicine.

The establishment of the National Health and Medical Research Council is a novel experiment. As Dr. Cumpston said in his inaugural address at the first session: "In no other country has an attempt been made to combine the preventive and curative aspects of medicine and the search for new knowledge in the science of medicine in one comprehensive scheme." The combination of national health with medical research necessitates a large council, whereas the allocation of money for research is obviously best done by a small body. The establishment of a committee of reference to deal with the coordination and the subsidizing of research is sound, and if the council leaves these matters in its hands, much of the objection which has been voiced in some quarters to the unduly high representation of State medicine on the council is met.

What now should be the policy which should guide the future development of research in Australia? In the first place it is vital to strengthen the existing university medical schools, now four in number, since Brisbane has a school founded in 1936. The emoluments of the chairs, not only in pre-clinical, but in the fundamental subjects of the first year, must be sufficient to attract men of the highest abilities, either from among our own graduates or from overseas. It is, however, idle to obtain men of talent as professors unless an adequate staff of lecturers and demonstrators is available to make the burden of teaching light enough to enable both the professors and their associates to carry out original work. Furthermore, money must be available for first-rate technical assistance and for apparatus and materials. The development of medical and surgical chairs should further the advance of clinical science. One of the most serious disadvantages under which the occupants of university chairs labour is the increased amount of time taken up by committees and faculties. This can be dealt with only by the appointment of a whole time executive officer to the medical school—a full-time dean whose functions should be wholly administrative.

Full use should be made of the existing institutes of research, none of which has sufficient income. The directors of such institutes should be freed from the burden of finding money for their research programmes, so that they might have more time for their own researches.

The National Health and Medical Research Council has in view the finding and training of young men and women for the future and the provision of opportunity so that our young graduates, who have the ability and desire to devote themselves to research, will not be compelled to seek an outlet for their energies in other countries. In the training of our future leaders in research we may well profit by the example of the United States of America, which has obtained from all over the world, irrespective of nationality, the ablest available people for university chairs and research positions, with the result that now, after the lapse of forty or fifty years, she is able to find among her own people enough brilliant and well-trained men to meet her needs. This introduction of new scientific blood is a necessary stage in the development of research in a country, and however much we may desire to see Australians in our picked positions, it is far more important for us to obtain the best people available, until such time as we can ourselves provide men of the very highest quality for our university chairs and other appointments.

From this necessarily incomplete story of the advance of medical science in Australia we may draw one certain inference, namely, that progress in the future will take place at an ever-increasing rate. It was Sir Richard Stawell's practice in teaching to draw the attention of his students to the unsolved problems of medicine. When they had worried sufficiently long without result over such a question, he would say: "I don't know; nobody knows." Though new discoveries give us fresh horizons, there is a steadily decreasing gap between the question asked by the clinician and its answer, and as time goes on less and less frequently will he be compelled to say: "I don't know; nobody knows."

AN ATTEMPT TO IMMUNIZE LAMBS AGAINST HYDATID DISEASE.

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Experimental Infestation of Dogs with Tænia Granulosus (Tænia Echinococcus).

So that a plentiful supply of fresh *Tænia granulosus* ova would be available for experimental purposes, two dogs were fed with fresh hydatid cysts obtained from sheep.

The results of this experimental feeding are described because other workers have found that the parasite grows at different rates in the intestine of the hosts. Leuckart[1] discovered mature worms seven weeks after feeding animals on hydatid material. Zenker, eleven weeks after feeding, found the parasites developed as regards size but still without eggs. Twenty-seven days after feeding von Siebold recognized hard-shelled eggs in the last joint. Southwell[2] noted that the fox harboured mature but not ripe parasites twenty-seven days after feeding, and the cat immature worms sixty-eight days after feeding. Stephens[3] stated that *Tænia echinococcus* took one to three months to grow. Perroncito[4] observed that proglottides formed twenty-four days after a dog had been fed with hydatid material.

Experiment.

The two dogs (*A* and *B*) used were about five months old, cross-breds resembling large-sized fox terriers. Several examinations of their fæces disclosed no cestode ova.

On June 5, 15 and 16, 1936, they were each given large feedings of sheep's livers containing numerous live hydatid cysts. On June 23, 1936, after being kept without water for two days, both dogs drank a large volume of hydatid fluid, rich in active hydatid sand. The fæces were examined microscopically at intervals of approximately ten days, and on September 1, 1936 (that is, twelve weeks and four days after the first feeding) a few mature ova of *Tænia echinococcus* were detected in the excreta of dog *A*. On September 10, 1936 (that is, thirteen weeks and six days after the first feeding), the fæces of dog *A* contained many hundreds of ripe segments, approximately three millimetres long by one millimetre wide.

From then on the fæces of both the dogs were examined macroscopically almost daily, but only on three or four occasions were any segments noticed, and then only in very small numbers. The second dog never discharged a large shower of ripe segments, nor was the performance repeated by the first dog during the ten months' period of observation. During this period *Tænia echinococcus* ova in the stools could be detected in approximately 50% of the untreated stools examined.

On February 4, 1937, no ova were detected after an examination for fifteen minutes of direct smears and of smears made after an attempt to concentrate any ova from the fæces by flotation in saturated brine. On February 5, 1937 (that is, a little more than five months since ova were first detected in the fæces), both dogs were given 8·0 cubic centimetres (three-quarters of an ounce) of castor oil. The bowels opened several times and the motions contained many ova and ripe segments. The *Tænia echinococcus* ova examined (see Figure I) were very similar in appearance to those of *Tænia saginata*. The diameter of the embryophore was almost constant at 41μ × 34μ. The external shell, on the other hand, varied considerably in diameter, but was usually 90μ × 75μ. Professor H. R. Dew[5] has stated that the average size of these ova is 33μ × 28μ.

On April 7, 1937 (that is, almost ten months after feeding the dogs), dog *A* was killed and a *post mortem* examination was made. The intestine was removed and opened along its whole length and the fæcal matter was carefully washed away under the running tap. The upper portion of the small intestine was free of *Tænia echinococcus*, whereas the lower half, except for the lowermost 20·0 centimetres (eight inches), was thickly covered with these parasites (see Figure II). There were literally thousands of parasites. The relatively large and opaque ripe segment would move about like seaweed under water. The heads were firmly anchored and some would remain attached to the mucosa and protected by the villi, the worms breaking if attempts were made to pull them off. However, large numbers of complete worms would fall off when the intestine was waved about vigorously in water. In this way the specimen for Figure III was prepared.

Remarks.

Because our dogs had several feedings of hydatid cysts, we are not in a position to give exactly the time which elapses before ripe segments are excreted with the stools. In one dog they were passed in great numbers thirteen weeks and six days after the first feeding and eleven weeks two days after the last.

It is strange that the passage of a shower of segments referred to was not repeated by the dog in question nor imitated in any way by the second dog. As both dogs at *post mortem* examination were shown to be heavily infested, it can be said that the clinical history is not uniform.

The most important observation is the difficulty experienced in detecting ova in the fæces of dogs, even when they are extremely heavily infested. This shows that the chance of detecting ova in the fæces of lightly infested dogs is extremely small, although such dogs are a real danger to the community. I infer that a veterinarian is making a serious mistake when he declares a dog free of infestation solely on the grounds that after purgation or anthelmintic medication he is unable to detect the parasites or their ova.

To obtain supplies of ova for experimental purposes the best method, in my opinion, is to infest a dog heavily by keeping it without water for two days, then giving it hydatid fluid rich in active hydatid sand to drink, and killing it three or more months later. A large number of complete parasites can then readily be obtained (see Figure III). Working on the advice of a leading research veterinarian in Melbourne, I was unfortunate enough to find myself picking out mature segments from the fæces passed after a dose of castor oil.

It might be mentioned here that Turner, Berberian and Dennis[6] claim to have produced in dogs a marked resistance to infestation with *Tænia echinococcus* by injecting them with a 1% suspension of scolices and germinative membrane obtained from hydatid in cattle. They observed similar results when they used scolices and germinative and cuticular membrane as the antigen. The antigens were prepared by drying the materials mentioned at 37° C., powdering and suspending them in 0·5% phenol solution.

Immunity to Hydatid Disease.

Miller and Massey's[7] observation of acquired immunity to *Cysticercus faciolaris* in rats, and our demonstration[8] of immunity in cattle to *Cysticercus bovis* immediately suggest that immunity to other cestodes is well worth serious investigation.

Attempts to Immunize Man and Animals against Hydatid Disease.

Professor Dévé, of Rouen, a great authority on hydatidosis, has recently performed immunity experiments and failed, as he anticipated, to demonstrate any immunity, either preventive or curative.

Ymas Apphatie[9] (1932) has claimed that hydatid anatoxin will immunize man against hydatid and that with his anatoxin he has produced retrogressive changes in cysts in two patients.

Dévé[10] (1934) performed three experiments, two with mice and one with rabbits, to disprove Apphatie's claim, and concluded that the hydatid anatoxin used by Apphatie had no curative power.

Dévé[11] (1933) was unable to prevent or cure hydatid in mice by using serum from street dogs. He considered that the serum might possess non-specific anti-parasitic properties. The hydatid was produced in mice by the intraperitoneal injection of hydatid sand.

More recently Professor Dévé[12] (1934) attempted to immunize four white mice by subcutaneous injections of a suspension of hydatid membrane obtained from the liver of sheep. He tested for any possible immunity by injecting scolices intraperitoneally. The scolices, however, developed into hydatid cysts.

Using the same antigen, he gave a series of subcutaneous injections to a rabbit bearing four obvious cysts from one and a half to two years old. The day after the last injection of antigen he injected hydatid sand subcutaneously. When the rabbit was killed some time later the four primary cysts were still alive and increasing in size, and secondary cysts, which had developed from the inoculation of hydatid sand, had reached the size of "half a kidney bean". Here again no immunity had been demonstrated.

A second rabbit was inoculated subcutaneously with hydatid sand, and immediately a series of subcutaneous inoculations of ground hydatid membrane was commenced. When these were completed, hydatid sand was again injected subcutaneously. Four months later cysts were found where both injections of sand had been given.

Professor Dévé's conclusion from these last three experiments was that hydatid membrane did not produce any immunity, either preventive or curative. That portion of the conclusion concerning preventive immunity is open to the objection that he tested for any possible acquired immunity by injecting scolices either intraperitoneally or subcutaneously. He may have produced immunity, but he did not demonstrate any. We consider that a more delicate and suitable test for immunity is to administer the ova of *Tænia echinococcus* by mouth, as this is the natural means of primary infestation, and in all probability oncospheres being conveyed by the blood stream would be more vulnerable than would scolices injected intraperitoneally or subcutaneously.

Dévé's reason for using mice and rabbits in his experiments, and scolices injected subcutaneously or intraperitoneally as a means of testing for any possible immunity, was apparently that he knew that by this means he could be certain of producing experimental hydatidosis. By the oral administration of *Tænia echinococcus* ova it is apparently difficult to produce hydatid regularly in any particular animal except the pig and the squirrel. Dévé[11] has pointed out the difficulty with white mice, and Haubner and Leuckart[13] have recorded it with lambs, sheep and goats.

In spite of these observations we decided to use lambs in our experiments, because they are the main intermediate host, and to give *Tænia echinococcus* ova orally, as this is the natural means of primary infestation. Our attempt to demonstrate the presence of immunity by this method was unsuccessful, but we did produce hydatidosis experimentally in lambs.

Experiment.

Eight lambs, born on or about June 15, 1936, were used. Four were kept as controls. On July 14, 1936, when the remaining four were four weeks old, they were each given the first of a series of eleven injections of hydatid antigen. These injections were completed on August 24, 1936. The hydatid antigen consisted of a suspension of scolices and ground hydatid membrane in hydatid fluid. Each of the four lambs received 66 cubic centimetres of this suspension. Five weeks after the last injection (September 29, 1936) the eight lambs were each given orally (by means of a rubber tube passed low down into the œsophagus) 100,000 fresh *Tænia echinococcus* ova suspended in water. Up to this time great care had been taken by hand-feeding the lambs to rear them

in a parasite-free environment. On February 5, 1937 (that is, eighteen and a half weeks later), two of the control lambs and two of the inoculated lambs were slaughtered. To our disappointment, although the whole of each carcass was carefully examined, no hydatid cysts could be recognized with certainty in any of the four animals.

However, the livers and to a less extent the lungs of the animals contained a great number of whitish lesions resembling miliary tubercles and about 1·5 millimetres in diameter. Probably the majority were to be found on the surfaces of the livers, and many contained a minute transparent cyst partially filled with whitish contents. Under the microscope the lesions were seen to be cystic, the wall of the cyst being composed of a few layers of young fibrous tissue lined internally by a delicate membrane containing occasional nuclei. The cyst was partially filled by an amorphous calcareous deposit. External to the fibrous tissue capsule was an area of marked cellular reaction wider than the cyst itself. The cells were mainly small lymphocytes; but there were a moderate number of eosinophile polymorphonuclear cells and a small number of giant cells.

From subsequent observations these lesions were in all probability young hydatid cysts; but they could also have been some other young bladder worm, especially *Cysticercus tenuicollis*, the larval stage of *Tænia marginata* of the dog.

It is interesting to note that Leuckart,[13] after feeding ripe proglottides of *Tænia echinococcus* to lambs, sheep and goats, made observations almost identical with those just described.

The remaining four lambs were killed and examined on June 11, 1937, that is, almost nine months after being artificially infested with eggs. The livers and to a less degree the lungs of all four animals contained a moderate number of cysts, varying in diameter from two to five millimetres. On close examination there appeared no doubt that the lesions were young sterile hydatid cysts. Actually there was a larger number of cysts in the inoculated animals than there was in the controls, so that there is no doubt that no appreciable immunity was produced by the antigen used.

Summary and Conclusions.

1. Two dogs were very heavily infested with *Tænia echinococcus*, and from the results of the examinations of their fæces for ova it is inferred that this is not a satisfactory method for detecting light infestations.

2. The diameter of the embryophore of *Tænia echinococcus* ova is almost constant at $41\mu \times 34\mu$, whereas the external shell varies in diameter, but usually measures $90\mu \times 75\mu$.

3. The important literature on immunity experiments on animals is briefly summarized.

4. Hydatid cysts were produced experimentally in lambs by feeding them with *Tænia echinococcus* ova. These cysts, when nine months old, were sterile and measured two to five millimetres in diameter.

5. No immunity sufficient to protect against infestation was produced in four lambs each inoculated with a total of 66 cubic centimetres of antigen consisting of ground hydatid membrane suspended in carbolized hydatid fluid.

Acknowledgements.

I am indebted to the trustees of the Baker Institute for providing facilities for the work, and I wish to express my thanks to the director, Dr. W. J. Penfold, for help of a general nature, and to Miss M. Phillips, B.Sc., who assisted me in collecting ova, preparing the antigen and in searching through the literature. My appreciation is also due to the Melbourne and Metropolitan Board of Works and its farm manager, Mr. F. Vincent, for looking after the lambs for several months on the board's farm. For the photography I am indebted to Mr. Lewis Booth.

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INDICATIONS FOR SPLENECTOMY IN PÆDIATRIC PRACTICE.[1]

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IN 1854 Henry Gray stated that the spleen played a part in blood production, regulated the quantity and quality of the blood, and acted "under certain circumstances as a reservoir for blood". It seems that all the splenic studies of the intervening eighty years have added very little to Gray's dicta.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on November 25, 1937.

Functions of the Spleen.

Of the many functions which have been attributed to the spleen, the following might be accepted as valid.

1. *The Reservoir Function.*—The splenic pulp forms a large backwater through which the blood circulates very slowly; this enables the spleen to play an important and useful part in regulating the blood flow to meet varying bodily demands. It is thought that perversions of this reservoir function may occur and may produce the "crises of deglobulization" described by Widal and others.

2. *The Formation of Blood.*—During intrauterine life the production of blood cells is a normal part of splenic function. After birth these areas of hæmatopoiesis subside rapidly; but they can be quickly revived during early infancy if there is sufficient demand. Apparently the spleen continues to produce lymphocytes and monocytes throughout life.

3. *Destruction of Blood.*—In the normal individual the spleen plays an important part in the disintegration and final disposal of red cells and the resultant production of bilirubin. Between 600 and 1,000 million red cells are destroyed each day by disintegration and fragmentation; the spleen acts as a graveyard for these worn-out cells and disposes of their final fragments.

The spleen's part in what might be called "pathological hæmolysis" is a very different matter and remains a most confused problem at present.

4. *Destruction of Blood Platelets.*—Possibly the blood platelets are also destroyed in the spleen. It has been suggested: (i) That the spleen actually destroys the platelets or removes them from the circulation by phagocytosis. This hypothesis seems doubtful in view of the fact that counts of platelets leaving and entering the spleen have shown very little variation. (ii) That a disturbance of some regulatory influence of the spleen over the megakaryocytes in the bone morrow results in a fall in platelet production. Unfortunately it has not been definitely established that the platelets arise from the megakaryocytes.

5. *Hormonal Action on Bone Marrow.*—Contradictory theories have been advanced as to the influence of the spleen on marrow function; possibly we might assume a normal stimulant and a pathological depressant factor.

6. *Defence of the Body.*—With the rest of the reticulo-endothelial system, the spleen plays an important part in the defence processes of the body.

It seems that the spleen is not essential for life, and that after splenectomy many of its functions are probably carried out vicariously by other representatives of the reticulo-endothelial system.

The most obvious effects of splenectomy in normal animals are: (*a*) a moderate hypochromic anæmia, which does not persist for more than two or three months; (*b*) a relatively sudden increase in the number of blood platelets (an increase varying from 50% to 200%), followed by a gradual fall to normal level after about twelve months; (*c*) a considerable increase in the total number of white cells (more particularly the granular forms), which persists for many months; (*d*) a proliferation of other parts of the reticulo-endothelial system.

Some workers have contended that the animal's red cells were more resistant to saline solution after splenectomy than before, and have supported their contention that the spleen has a "weakening effect" on the erythrocytes by showing that the red cells in the splenic vein are less resistant than the entering cells.

With your permission, I propose to offer a brief and rather dogmatic tabulation of the main indications for splenectomy and to reserve any detailed discussion for the two most common indications, namely: (*a*) essential thrombocytopenic purpura, and (*b*) certain forms of hæmolytic anæmia (including acholuric jaundice).

Indications for Splenectomy.

The literature contains reports of splenectomies performed during childhood for many surprising conditions varying from primary polycythæmia and Niemann-Pick's disease to *kala-azar* and acute rheumatism. At the present time the possible indications for splenectomy during infancy or childhood might be classified as follows: (i) injury to the spleen; (ii) splenic tumours; (iii) Gaucher's disease; (iv) Banti's syndrome; (v) thrombosis of the splenic vein (Wallgren) (vi) essential thrombocytopenic purpura; (vii) certain forms of hæmolytic (erythronoclastic) anæmia, including acholuric jaundice.

The blood platelet level is frequently low in Gaucher's disease, and splenectomy may be of definite value in this respect. In the absence of thrombocytopenia, splenectomy may relieve the discomfort caused by the large size of the spleen, but obviously does not offer any cure for the disease itself. Splenectomy does not prevent the occurrence of bone involvement, and it is possible that it may precipitate early skeletal infiltration.

Attempts to classify certain types of unexplained anæmia and splenomegaly of childhood under the title of Banti's syndrome have resulted in much confusion and contradiction. Poynton, Thursfield and Paterson doubted whether Banti's syndrome ever occurred before puberty, and could recall very few cases in which subsequent events upheld the diagnosis. Leonard Parsons agreed that the earlier stages of Banti's syndrome might be recognized in childhood, after careful exclusion of other possibilities.

Theoretically, if all other ætiological factors could be excluded, anæmia, leucopenia and splenomegaly might be accepted as the early signs of Banti's syndrome. Unfortunately, in practice it is very difficult to exclude all other ætiological possibilities, more particularly vascular abnormalities of the portal system (such as primary or secondary thrombosis of the splenic vein).

Splenectomy in the earlier stages usually produces a pronounced improvement in the condition; but

occasionally post-operative improvement is followed by a relapse within a year or more. There may be some increased risk of intravascular clotting as a result of the unduly high platelet counts which frequently follow splenectomy, but practically this risk seems very slight.

Thrombosis of the splenic vein may simulate Banti's disease, but is usually associated with a leucocytosis. According to many observers, splenectomy is a successful form of treatment in the majority of cases; but Wallgren points out that the results are very variable, and that there is a risk of further thromboses.

Essential Thrombocytopenic Purpura.

It seems probable that platelet production may be depressed by certain lesions of the bone marrow, and it is most important that such lesions should be carefully excluded before a diagnosis of essential thrombocytopenic purpura is made. Symptomatic thrombocytopenic purpura may be the result of (*a*) other blood dyscrasias (more particularly the leuchæmias and aplastic or myelophthisic anæmias), (*b*) sepsis, (*c*) drug intoxication, (*d*) irradiation, (*e*) Gaucher's disease.

Essential thrombocytopenic purpura appears to be a primary disease, and usually occurs suddenly in a healthy child. There is rarely any family history of purpura[1] or other blood dyscrasia, and there is usually a characteristic absence of any other signs of ill-health, apart from the ill-effects of the hæmorrhages. The other classical features of this syndrome are the following:

1. *Thrombocytopenia.*—The platelets usually fall to less than 30,000 per cubic millimetre. The critical platelet level for hæmorrhagic lesions probably lies between 30,000 and 40,000 per cubic millimetre; but there seems to be an unfortunate discrepancy between platelet level and hæmorrhagic tendency.

2. *Other Blood Changes.*—The bleeding time is increased from the normal period of three minutes to more than ten or even twenty minutes. The coagulation time is normal, but clot retractility is defective.

3. *Purpuric and Hæmorrhagic Manifestations.*—Purpuric lesions, petechiæ and ecchymoses are especially evident on the legs or on any part of the body exposed to trauma. As would be expected, the capillary resistance test is always positive, but it is interesting to note the apparent lack of relationship between this test and the level of the platelet count. Hæmorrhages from mucous membranes are relatively frequent and intracranial hæmorrhage has been stressed as an important hazard of this disease in early life. Occasionally, affected children have suffered gangrene of one or more extremities.

4. *Splenic Enlargement.*—A variable degree of splenic enlargement is often found, but is by no means a constant feature of this syndrome. In many children this enlargement seems more evident during the active stage of the purpura.

The duration of this disease varies from a few days to many years, and all gradations of severity may be encountered. In many of the acute cases there is only one attack, while in the chronic relapsing forms attacks may occur at intervals of one to six months, each attack persisting for days or even weeks.

For the purposes of this present discussion, cases of essential thrombocytopenic purpura might be subdivided into the following groups:

1. Acute.
   - (*a*) Benign. (A very large group—usually spontaneous recovery and frequently only one attack.)
   - (*b*) Fulminant. (A small group—often fatal. If the child survives there is rarely any recurrence of the disease.)
2. Chronic.
   - (*a*) Relapsing or intermittent. (Platelets may be normal between attacks.)
   - (*b*) Continuous. (Low platelet count is constant, though clinical manifestations of the disease are not always present.)

Splenectomy.

In view of the present confusion as to the relationship between the spleen and platelet destruction, it seems surprising that the first splenectomy for the relief of thrombocytopenia was performed about twenty years ago. In 1915 Frank, of Breslau, maintained that the spleen inhibited the formation of platelets and suggested its removal in thrombocytopenic purpura. A year later Kaznelson, of Prague, was convinced that the spleen destroyed the blood platelets, and proceeded to remove it with success in a case of thrombocytopenic purpura.

During the next decade there was a tendency to perform the operation of splenectomy in the majority of patients who had suffered more than one attack of essential thrombocytopenic purpura. In recent years it has been realized that in most of the acute cases (probably three out of four or five) the patients tend to make a complete and permanent recovery with or without treatment, and that many of the children attacked never have a second attack. Consequently the present tendency is to reserve splenectomy for those severe chronic types of essential thrombocytopenic purpura which persist despite frequent blood transfusions and other medical treatment. In the light of our present clinical and experimental knowledge of this subject, it must be admitted that splenectomy does not remove the fundamental cause of essential thrombocytopenic purpura; if we appreciate this fact and assume that splenectomy merely means the removal of an important site for platelet destruction, then the interpretation of the immediate and remote results of this operation becomes easier.

An increase in the number of platelets is the most constant change in the "normal" child's blood picture after splenectomy. Two to five days after the operation there is usually a rapid rise in the

[1] Instances of hereditary and familial forms have been recorded, and Brown and Elliott report the history of a woman who became pregnant after splenectomy and gave birth to a child with classical essential thrombocytopenic purpura. The familial form of purpura which is best known is characterized by a normal platelet count.

platelet count; a peak of perhaps 800,000 per cubic millimetre is reached at the end of seven to ten days, and may persist for two or three weeks before a slow fall to the normal level begins. This slight delay in the original post-operative platelet rise has been demonstrated in cases of traumatic rupture of the spleen, and may be contrasted with the immediate post-operative rise of platelets which usually occurs in thrombocytopenic conditions.

Steiner and Gunn have shown that in animals the increase in blood platelets following splenectomy is not greater or more prolonged than that which follows other equivalent degrees of surgical trauma; but practical experience suggests that splenectomy produces a higher and more prolonged platelet response in children that other forms of surgical trauma.

As a rule, the results of "interval" splenectomy in cases of chronic essential thrombocytopenic purpura are good, failures are rare, and recurrences of hæmorrhages are relatively infrequent. Complete failure to show any increase in platelets after splenectomy may occur in rare cases, and has suggested the possibility that some of these children may be suffering from inadequate or imperfect platelet formation. Marrow smears have been examined in an attempt to exclude such a possibility, but must be of doubtful value until we have more definite proof that the platelets arise from the megakaryocytes. Occasional reports of hæmorrhages occurring three or four years after splenectomy may suggest the theoretical possibility of hyperplasia of supernumerary spleens or other portions of the reticuloendothelial system; but Witts has pointed out that "if we compare the frequency of relapse in idiopathic thrombocytopenic purpura with its rarity in acholuric jaundice, we must doubt whether growth of a spleniculus is a common explanation of relapse".

The following case history illustrates the response to splenectomy of a patient suffering from essential thrombocytopenic purpura.

A.W., aged thirteen years, was admitted to the Royal Alexandra Hospital for Children in November, 1928, with a history of frequent epistaxis and many unexplained bruises during the previous two years. There was no family history suggestive of blood dyscrasia or other significant disease. At the time of his admission to hospital this boy was suffering from a severe epistaxis, a large ecchymosis of his right thigh and many bruises on his body and limbs. At this time examination of his blood revealed a pronounced hypochromic anæmia; his blood platelets were too sparse for any accurate count, and his spleen was slightly enlarged. He was given some intramuscular injections of sterile milk without any platelet response being produced, and later he received a blood transfusion. Following the transfusion his blood platelets showed a very slight increase in number (reaching approximately 60,000 per cubic millimetre), he seemed better and remained free of epistaxis for about six weeks. After this intermission, several severe attacks of epistaxis occurred, necessitating a further blood transfusion. After this transfusion he was relatively free from hæmorrhages for nearly three months, but was readmitted to hospital in June, 1929 (that is, seven months after his original admission), suffering from severe epistaxis and many bruises on his limbs. No platelets were seen in his blood films; he was given a transfusion, and two days later splenectomy was performed by Dr. Hipsley. His blood platelets had reached 186,000 within ten days of the operation, and twelve weeks later he was apparently well, his blood platelets totalled 180,000 per cubic millimetre, and he was free from all evidences of purpura. At the present time he is twenty-two years old, in excellent health and earning his living as a timber-getter. (See Figure I.)

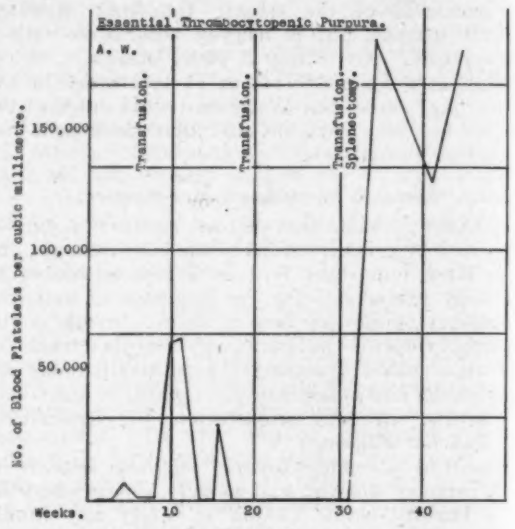

FIGURE I.

As recurrences of some severity, including severe uterine hæmorrhages, may occur suddenly with the onset of menstruation, it is wise to consider the question of splenectomy before puberty in relatively mild chronic types occurring in girls.

If splenectomy is indicated, the operation should be performed preferably during a period of temporary remission, and preliminary blood transfusions should be given with the object of raising the red cell count to at least 3,500,000 per cubic millimetre, if possible. The medical attendant who is faced with the treatment of one of the more prolonged relapses of essential thrombocytopenic purpura may need much confident patience to help him in his decision to persist with blood transfusions and other medical measures until a remission occurs. His decision on this course of action should be strengthened by the knowledge that this disease is essentially one of relapses and remissions, that remission from even the most severe relapse is the rule rather than the exception, and that the dangers of splenectomy are greater during a period of relapse.

The platelet level after splenectomy usually rises sharply within one or two hours of the operation, reaches a variable maximum within the next seven to ten days, and then falls slowly to a slightly lower and more constant level. Theoretically, it might be supposed that there was a risk of thrombosis in those cases in which an unduly high platelet response is seen after splenectomy, but, practically, this seems to be a very rare complication.

I have avoided any discussion of other methods of treatment (for example, ligation of the splenic artery, irradiation of the splenic area, use of snake venoms, injection of various protein substances and administration of ascorbic acid) as being outside the scope of this paper.

The Hæmolytic (Erythronoclastic) Anæmias of Infancy and Childhood.

Although I have used the word "hæmolytic" throughout this paper, I much prefer the more correct, if less euphonious, adjective "erythronoclastic", which emphasizes the fact that in many cases there is not only destruction of the circulating blood, but of the whole erythron.

Before I discuss the value of splenectomy in this group of blood dyscrasias, it is essential that an attempt to understand and classify them on an ætiological basis should be made. The following classification is based on the work of Parsons, Hawksley and Gittins (1933), and has proved very helpful to me in the study and understanding of these dyscrasias.

1. Hæmolytic anæmias of the new-born. *Icterus gravis* and the acute hæmolytic anæmias of the new-born (with or without erythroblastosis). In certain of the acute hæmolytic anæmias of the new-born the advisability of splenectomy might be considered, if excessive hæmolysis has continued despite repeated blood transfusions. I understand that Cooley has seen recovery follow splenectomy in two or more cases of this type.
2. Hæmolytic anæmias of infancy and childhood.
   (*a*) Acute hæmolytic anæmia (Lederer type). These cases usually react dramatically to blood transfusion, and splenectomy is not indicated.
   (*b*) Subacute and chronic hæmolytic anæmia (including von Jaksch's syndrome and possibly Cooley's erythroblastic anæmia).
3. Congenital anomalies of the erythron.
   (*a*) Acholuric jaundice.
   (*b*) Sickle cell anæmia.

If we find that a child is suffering from an anæmia associated with a positive Van den Bergh reaction[1] and has an excess of urobilin or urobilinogen in his urine, we may assume that he is probably suffering from hæmolytic anæmia, and it is our primary duty to exclude acholuric jaundice and such causes as infection, syphilis, nephritis, lead and other poisons. Further, the degree and type of his marrow response should give us some indication of the amount of damage suffered by his marrow, and may help us to assess the balance between hæmatopoiesis and hæmolysis.

We may recognize an ascending series of gradations, from the acute hæmolytic anæmias through the various subacute and chronic forms to the classical syndrome of familial acholuric jaundice, which seems to represent the most chronic type of hæmolytic anæmia. As a rule, the fulminating acute hæmolytic anæmia of the Lederer type responds dramatically to transfusion, and splenectomy usually relieves patients suffering from acholuric jaundice; but there seems to be no certain therapy for the atypical intervening types, which show a very variable uncertainty in their response to splenectomy and other forms of treatment.

[1] Obviously there may be no response to the Van den Bergh test under certain conditions in cases of hæmolytic anæmia, and for this reason a positive reaction must not be regarded as an essential and constant feature of these anæmias. Further, it seems reasonable to follow the present-day tendency to regard the Van den Bergh reaction as quantitative rather than qualitative, and to accept an immediate positive reaction as evidence of a high icterus index, a delayed or indirect reaction as evidence of a low icterus index, and the biphasic reaction as representing an intermediate level of this index.

Subacute Hæmolytic Anæmia.

Subacute hæmolytic anæmia is a relatively rare condition, but one in which splenectomy may be of therapeutic value. Whitby and Britton do not offer any separate description for this group, and Parsons and Hawksley (1933) stated that they had seen only four cases of this type.

The following details of a case which came under my care early this year will serve to illustrate this syndrome and its occasional dramatic response to splenectomy.[1]

I.H., aged twenty-one months, was referred to me by Dr. Dixon Hughes with a history of increasing pallor during the previous month or six weeks. There was no family history suggesting blood dyscrasia; his parents were Australian and his grandparents of British stock. His diet and progress during his early infancy had been satisfactory, and he had not suffered from any obvious illnesses. He was a pale but well-nourished child, whose skin and conjunctivæ appeared distinctly icteric. His clinical history and his blood picture, combined with the constant presence of a positive indirect Van den Bergh reaction and a persistent excess of urobilin in his urine, supported the provisional diagnosis of subacute hæmolytic anæmia. No obvious cause for this abnormal degree of hæmolysis was detected; his urine did not contain lead in pathological quantities, his blood gave no response to the Wassermann test, his red cells were slightly resistant to hæmolysis in serial dilutions of saline, and radiographic examination of his skull and long bones did not reveal any abnormalities except some slight rhachitic changes.

During a period of three months' observation this child failed to show any improvement in his blood picture, despite various forms of treatment, including five blood transfusions, and he continued to exhibit signs of excessive hæmolysis. Frequent examinations of the resistance of his red cells to serial dilutions of saline failed to reveal any increase in their degree of fragility. Histological examination of material obtained by sternal trephine suggested a fully active marrow. In view of these findings, it was decided that splenectomy should be carried out in the hope that removal of the spleen might diminish the amount of hæmolysis and so establish a better balance between red cell formation and red cell destruction.

Splenectomy was performed by Dr. Hipsley on July 27, 1937, and the child made a dramatic and uninterrupted recovery. Within three days of the removal of the spleen his blood picture had shown a marked improvement, he had lost any obvious icteric tinge, his serum gave no response to the Van den Bergh test, and he had all the appearances of a healthy child. (Figures II and III.)

It is obviously much too early to offer any exact opinion about this child's hæmatological future; but at the present time (four months after splenectomy) he appears to be very well, his blood picture is normal, and there is no evidence of excessive hæmolysis or of any increase in the fragility of his red cells.

[1] Full details of this case are reported by Dr. Phyllis Anderson in this number of the journal.

Chronic Hæmolytic Anæmia.

The results of splenectomy in the treatment of the chronic hæmolytic anæmias have been disappointing.

Von Jaksch's Anæmia.—Included amongst the chronic hæmolytic anæmias of infancy are a group of cases in which the marrow response is satisfactory, and which are recognized to have a relatively good prognosis despite their very slow course. Apparently the marrow has not been severely injured and the balance between hæmatopoiesis and hæmolysis is in the infant's favour. It is cases of this type (possibly associated with rickets or accompanied by widening of the medullary spaces of the

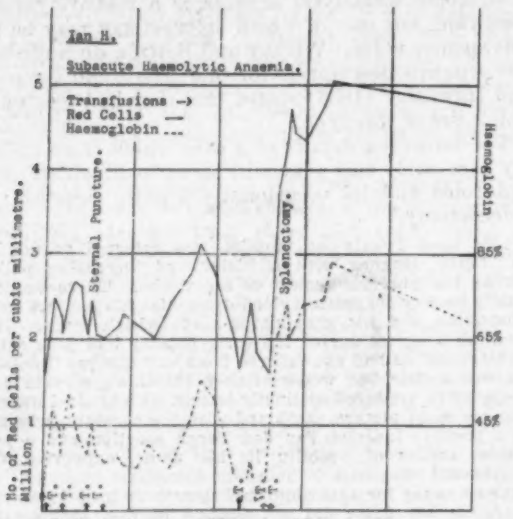

FIGURE II.

skull and long bones) which have been classified under the title of von Jaksch's anæmia. In view of the natural recovery of the majority of these patients, splenectomy is not indicated. Further, when splenectomy has been performed in such cases, the results have been disappointing.

Cooley's Erythroblastic Anæmia.—It is difficult to know how to classify Cooley's erythroblastic anæmia, for although hæmolysis is a constant feature, it is doubtful whether the disease is primarily hæmolytic in origin. For the sake of simplicity, I have included it under the title of chronic hæmolytic anæmia.

This disturbance, which is limited to families of Mediterranean origin, occurs during infancy and early childhood, and is characterized by splenomegaly, pronounced erythroblastosis, classical changes in the skull and long bones, and a somewhat mongoloid appearance. It should be emphasized that these changes in the skull and long bones, which are the direct result of rapid widening of the marrow spaces, are not pathognomonic of Cooley's anæmia, and have been reported in cases of acholuric jaundice, sickle cell anæmia and chronic hæmolytic anæmia.

Up to the present day no classical examples of this syndrome have been described in Australia; but it is possible that the steady influx of Mediterranean peoples to this country may alter the position.

Cooley states that splenectomy is of no value, and may grossly aggravate the anæmia. A curious and rapid increase in the number of nucleated red cells occurs after the operation. The constant and complete failure of splenectomy in the treatment of this condition seems to support Cooley's recent contention that, although hæmolysis is a constant feature, it is unlikely that the disease is primarily hæmolytic in origin.

Splenectomy for Chronic Hæmolytic Anæmia.—Shortly after Cooley's original description of chronic hæmolytic anæmia appeared in the literature, I was fortunate enough to observe a form of this syndrome in two brothers of Australian parentage, who did not exhibit erythroblastosis, but did show the clinical and radiographic skull changes and the mongoloid facial appearance described by Cooley.

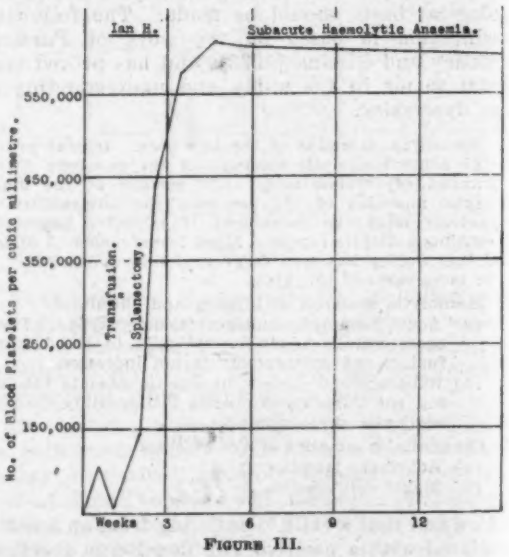

FIGURE III.

These two brothers, who were admitted to the Royal Alexandra Hospital for Children under Dr. Wade's care, had been pale and sallow since infancy, and had both suffered from mild attacks of jaundice. Another brother, who was reported to have suffered from splenomegaly and jaundice, had died of pneumonia. There was no further family history of jaundice, anæmia or splenomegaly. Both boys appeared definitely anæmic and slightly icteric; their faces suggested the mongoloid type, their spleens were enlarged, and their skulls showed the classical clinical and radiographic changes described by Cooley in his original paper. Their blood pictures, the persistently normal fragility of their red blood cells and the fact that the Van den Bergh test gave a delayed direct reaction in both children, all supported the conclusion that they were suffering from a hæmolytic anæmia. The red blood cells of the elder of these brothers (D.C.) ranged between 1,000,000 and 2,000,000 per cubic millimetre for more than eighteen months, during which period the fragility remained consistently normal and the serum yielded a positive indirect Van den Bergh reaction. At the end of this period of observation, splenectomy was performed by Dr. Wade. Following this operation there was a slight but definite improvement in the blood picture (see Figure IV); apparently some improvement in the blood picture was maintained for nearly a year, and the boy was able to return to his school in the country for several terms. This

boy died about three years after his discharge from hospital; unfortunately, no details of his final illness are available.

The blood picture of the younger of these brothers was approximately the same as that of his elder brother. This boy did not have his spleen removed, and is reported to be well at the present time.

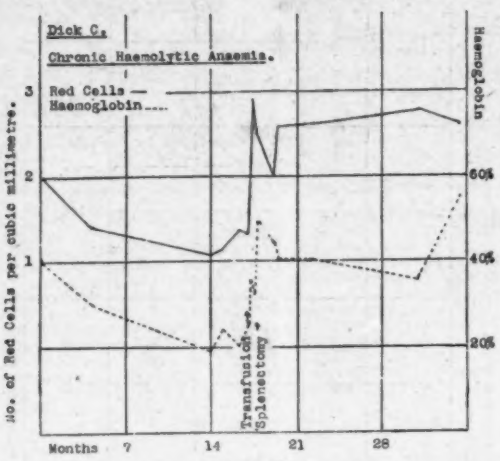

FIGURE IV.

The following history of a case of chronic hæmolytic anæmia is of particular interest because of the apparent absence of any hæmatological response to ligation of the splenic artery.

E.M., aged four years, was admitted to the Royal Alexandra Hospital for Children under Dr. Macintosh's care because of an increasing pallor of more than twelve months' duration. His diet and progress during infancy had been satisfactory, and there had been no obvious illnesses. There was no family history of blood dyscrasia or other significant disease. At the time of his admission to hospital his spleen was grossly enlarged, and his blood picture, combined with the presence of a positive indirect Van den Bergh reaction and an excess of urobilin in his urine, suggested a chronic hæmolytic anæmia. No obvious cause for this abnormal degree of hæmolysis was detected, and the fragility of his red cells was not increased. During the next ten weeks he was given three blood transfusions, and showed sufficient response to be allowed to leave hospital for a time. After a month at home he suffered a relapse and was readmitted to hospital for immediate blood transfusion. At this time the enlargement of his spleen was much more marked and his slightly icteric pallor more pronounced. During the next two weeks two more blood transfusions were given, and a week later ligation of the splenic artery was performed by Dr. Davis and another transfusion was given. During the five months which had preceded this ligation the fragility of the red cells had remained consistently normal, the blood platelets had been abundant, normoblasts and numerous reticulocytes had been seen in the blood films. Apparently ligation of the splenic artery did not produce any obvious change in these findings and did not control the hæmolysis (see Figure V). Finally, four months after this operation, his red cells and hæmoglobin value had fallen to levels similar to those found at the time of his admission to hospital. At this stage a blood transfusion was given and splenectomy was performed by Dr. Davis. Unfortunately, this operation proved very difficult on account of numerous vascular adhesions, and the child died about thirty-six hours later. A blood count, carried out twenty-four hours after the operation, showed the early evidences of an excellent response to the splenectomy (including a rise in his platelet count from 55,000 to 200,000 per cubic millimetre).

Congenital Anomalies of the Erythron.

Acholuric Jaundice.

Acholuric jaundice may be defined as a chronic hæmolytic anæmia characterized by crises of excessive blood destruction, a constantly high percentage of circulating reticulocytes, an enlarged spleen, increased fragility of the red cells and a variable amount of jaundice. The presence of the classical small spheroidal red cells seems to be a constant feature of the familial forms. The cause of acholuric jaundice is unknown, but it is assumed that it is the result of a congenital defect in the erythron.

Many authorities doubt whether an acquired type should be recognized, regard such cases as familial forms which have passed unrecognized through early life, and think that an exhaustive and competent examination would reveal an hereditary factor. From the point of view of prognosis, it is particularly helpful to recognize an acquired form of acholuric jaundice, and to realize that the more atypical examples of this form may be practically indistinguishable from the subacute and chronic hæmolytic anæmias.

1. *Familial Forms.*—The affected members of a family may exhibit extremely variable clinical pictures, varying from the child who in the course of a routine clinical examination is found to have

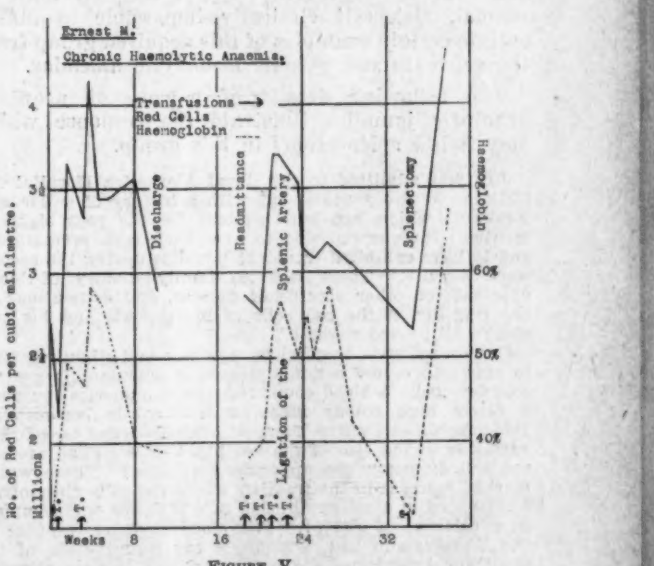

FIGURE V.

some slight splenomegaly, to the child who presents all the signs of the classical syndrome. Hæmolytic crises of varying degree may occur once or twice a year, or even more often. Many of the children seem to be comfortably adjusted to their low red cell count, and, as Chauffard has said, are "more jaundiced than sick"; but it must be remembered that these children have "an unstable present and possibly a perilous future", for their degree of anæmia depends on their ability to balance blood

destruction and blood production, and they are never free from the risk of a sudden hæmolytic crisis. Fortunately, the risk of a severe hæmolytic crisis appears to be slight in the familial forms; in fact, Parsons and others do not consider splenectomy essential in the milder examples of this group, and point out that the prognosis is naturally good in these cases.

For the remaining types of familial acholuric jaundice, "interval" splenectomy would seem to be essential, and both the immediate and remote results are most satisfactory. After splenectomy in such cases the excessive hæmolysis ceases, the blood picture improves, jaundice disappears, there is a rapid fall in the number of reticulocytes and the mean diameter of the red cells often increases; but there is little or no alteration in the undue fragility of the red cells.

2. *Acquired Forms.*—In this rather ill-defined group of cases the prognosis is somewhat graver, the anæmia is usually more pronounced, the number and severity of the crises seem to be greater, and the results of splenectomy are less certain. Further, certain children in this group seem to face the risk of a severe hæmolytic crisis after blood transfusion.

The fact that the spherocytosis, so characteristic of the familial forms, is a much less constant feature, and that the degree of red cell fragility may be normal, makes it clinically impossible to differentiate certain examples of this acquired group from the subacute and chronic hæmolytic anæmias.

The following details of a case of acquired acholuric jaundice illustrate the response which may follow splenectomy in this group.

J.H. was admitted to the Royal Alexandra Hospital for Children at five years of age with a history of pallor and weakness which had been evident for the past eighteen months. He was reported to have been born prematurely and to have exhibited transient jaundice during the second week of life. There was no family history of blood dyscrasia or other significant disease, and estimations of the fragility of the red cells of his parents and his two sisters all proved normal.

On admission to hospital he was pale and slightly icteric in appearance, and a minor degree of splenic enlargement was detected. A blood count revealed a numerical anæmia, a fairly high colour index, a neutrophile leucocytosis, evidence of an active marrow response, and a marked variation in the size of the red blood cells. (The average red cell diameter was approximately 5·3μ.) There was a marked increase in the fragility of the red cells (hæmolysis commenced in a saline dilution of 0·52% and was complete in a dilution of 0·45%). The blood gave no response to the Wassermann test, and an X ray examination of the skull and long bones did not reveal any obvious changes.

After a slight improvement in the blood picture during the first week, the count remained practically unaltered for the next month, and then showed evidence of a definite relapse.

Splenectomy was performed by Dr. Rogers about six weeks after the child's admission to hospital. The hæmatological response to the splenectomy is well illustrated in Figure VI. The blood changes are set out in Table I.

Sickle Cell Anæmia.

This severe hæmolytic anæmia is probably the result of a developmental defect of the erythron and is largely, but not entirely, confined to negroes. Most authorities are satisfied that splenectomy is of little avail in the treatment of this condition.

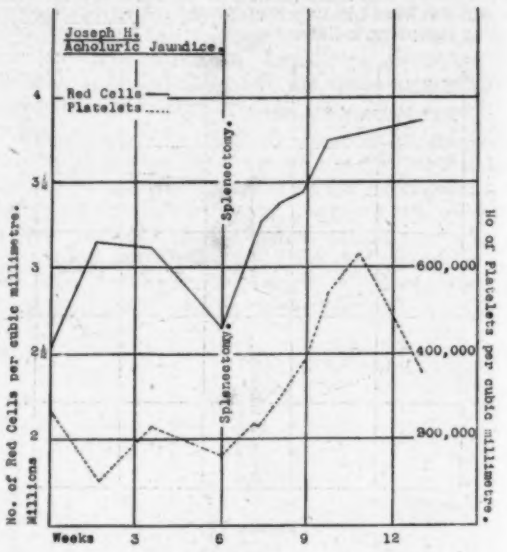

FIGURE VI.

TABLE I.

J.H.—Acholuric Jaundice.

Date.	Number of Red Cells per Cubic Millimetre.	Reticulocytes.	Hæmoglobin Value.	Number of Platelets per Cubic Millimetre.	Fragility.
		%	%		%
14/6/32	2,550,000	4	48	254,600	0·52–0·45
26/6/32	3,160,000	5	54	103,350	
8/7/32	3,140,000	4	53	230,000	
26/7/32	2,620,000	–	48	160,000	0·6–0·42
3/8/32		Splenectomy.			
4/8/32				235,000	
5/8/32	3,280,000	1	60	235,000	
8/8/32	3,380,000	Less than 1	62	290,000	0·6–0·42
12/8/32	3,430,000	Less than 1	67	380,000	0·6–0·42
18/8/32	3,740,000	Less than 1	68	530,000	0·6–0·36
26/8/32	3,750,000	Less than 1	70	635,000	0·6–0·36
12/9/32	3,860,000	Less than 1	72	350,000	

Acknowledgements.

In conclusion, I wish to thank the honorary medical staff of the Royal Alexandra Hospital for Children for permission to include details of their cases, and Dr. Phyllis Anderson for her help and criticism.

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Reports of Cases.

ANÆMIA CURED BY SPLENECTOMY.

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I.H., AGED twenty-one months, was referred by Dr. T. Dixon Hughes, and admitted to hospital on April 6, 1937. He was the first child of healthy Australian parents, in neither of whose family was there any history of jaundice or blood dyscrasia.

The boy had been breast fed and weaned onto a normal mixed diet; he was brought on account of pallor. The parents had noticed him becoming pale for three weeks, but they thought that it was four months since he had been quite normal.

On examination he was found to be a well grown child with no loss of subcutaneous fat; he had an icteric tinge to the skin and conjunctivæ. The spleen was not palpable, and there were no clinical signs of rickets. The symptoms were loss of appetite, listlessness and irritability.

A blood count revealed a severe anæmia, with 1·5 million red cells, 30% hæmoglobin, and 7,200 leucocytes per cubic millimetre. Stained films showed a considerable variation in size and staining of the red cells; the mean average diameter of the cells was 7·5μ (see Figure I). Two normoblasts were seen; the white cells were normal. The fragility test revealed a slight resistance of the red cells to hæmolysis in serial dilutions of saline solution; the serum gave an indirect positive reaction to the Van den Bergh test; there was a strongly positive reaction for urobilin in the urine. The blood gave no reaction to the Wassermann test. X ray examination of the long bones revealed slight rhachitic changes only.

A blood transfusion of eight ounces was given by the citrate method, and this was followed by a slight improvement, with a reticulocytosis of 2·2%. This was not maintained, however, the red cell count falling below two million per cubic millimetre in less than one week.

Iron therapy was instituted, and when an examination of the fasting gastric juice revealed the absence of free hydrochloric acid, this acid was added without improvement in the total count.

Examination of the child's urine for lead and a search for possible intake of lead in the drinking water of the home were made, but without success.

As the total red cells remained at a level of two million per cubic millimetre, another transfusion of six ounces was given, and then a depot dose of "Campolon" was given by intramuscular injection, and daily doses were also given. Daily reticulocyte counts were made, but there was still no response.

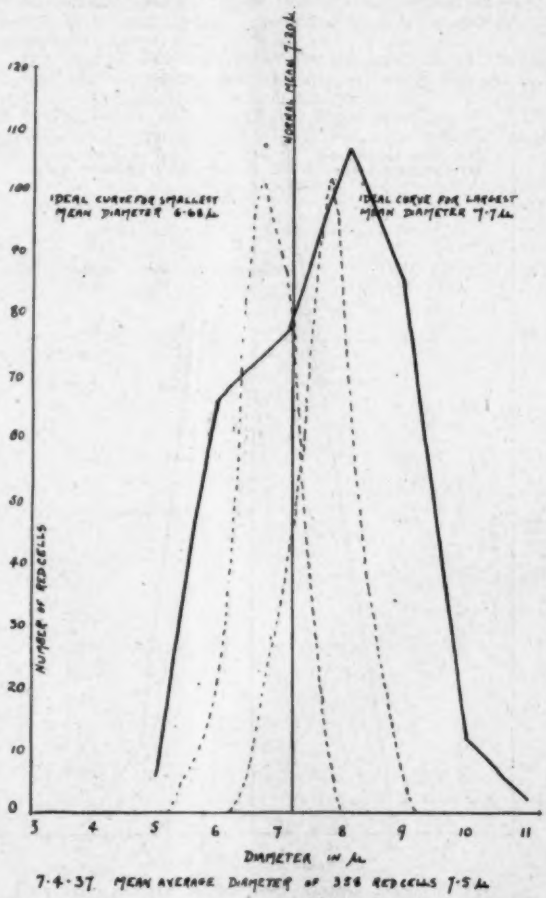

FIGURE I.

A sternal trephine was made on May 3 to investigate the condition of the marrow, and this was followed by another transfusion. A period of remission then set in, and the count climbed slowly to 3·3 million, with 55% hæmoglobin, and 6,700 leucocytes per cubic millimetre. At this time the child went home at the request of the parents.

He was readmitted to hospital on July 6, having had a relapse. A molar tooth had erupted, and the child had had an upper respiratory tract infection which had affected other members of the household. His skin was again very yellow, he was refusing food and was very listless. The blood count was 1·6 million, with a hæmoglobin value of 27%, and with 6,700 leucocytes and 46,000 platelets per cubic millimetre.

A further blood transfusion was given, and splenectomy was decided on, if the child's condition could be made

satisfactory for anæsthesia. Two transfusions were given three days apart, and the red count reached 4·3 million per cubic millimetre, and that of the platelets 146,000.

Splenectomy was performed on July 27 by Dr. P. L. Hipsley, and the child made an uninterrupted recovery. On July 28 the red cells had maintained their level, the platelets numbered 306,000 per cubic millimetre, and after one month the red cells numbered 5,050,000, with a hæmoglobin value of 77%, and the platelets numbered 610,000 per cubic millimetre. The mean average diameter of the red cells was 7·13μ (see Figure II). The serum gave no

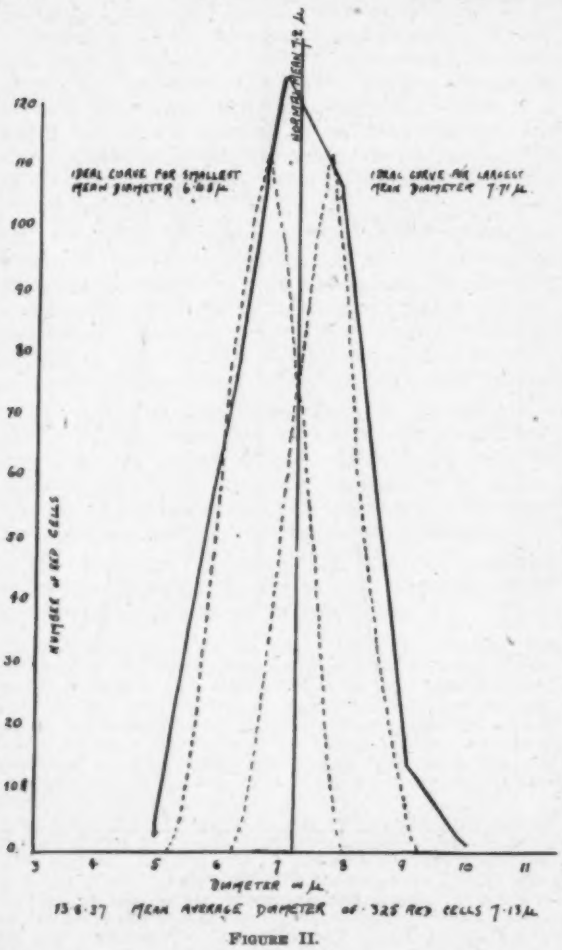

FIGURE II.

reaction to the Van den Bergh test, the skin and conjunctivæ were clear, and the child was well (see Figure III).

Histological Findings in the Marrow.—Smears showed the cells in the marrow to be well distributed, with many areas in which the red cells predominated, with normoblasts scattered through them. Other areas were composed chiefly of granular cells, the myelocytes being the most numerous; then came the mature neutrophile cells, while the blast cell was present in only small numbers. Eosinophile myelocytes were also seen.

The interstices of cortical bone were well filled, and there were areas chiefly occupied by red cells, others by white cells. In the red areas there were numbers of nucleated cells, some with pyknotic nuclei and ample hæmoglobin, others with bluish cytoplasm and open nuclei of the erythroblast type.

The myeloid areas were mainly occupied by myelocytes, a few of which had eosinophilic cytoplasm, and there were occasional megakaryocytes, the largest cells visible in the whole section, with wide pale cytoplasm and large lobed nuclei. The appearances were those of fully active marrow (see Figure IV).

Histological Findings in the Spleen.—The spleen was of normal outline, with three notches in the anterior border, and weighed 80 grammes. The weight of the new-born spleen lies between 8 and 20 grammes, and is said to double itself in the first year. The variations due to vascular change are enormous, and the patient's spleen cannot be said to be greatly enlarged.

On section the capsule was of normal thickness, the pulp appeared uniformly dark red, and it bled considerably after removal. Microscopic examination of sections revealed a normal capsule and inconspicuous trabeculæ. Malpighian bodies were large and numerous. Each had two zones, the

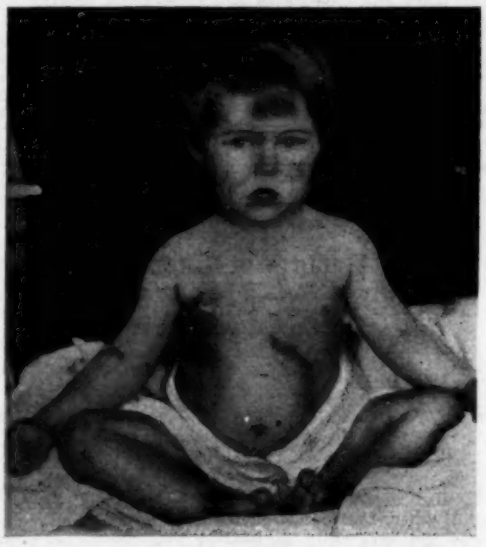

FIGURE III.

outer composed of lymphatic tissue, the small round cells with dark nuclei being typical lymphocytes. The central core was composed of loosely packed reticulum cells, with large pale nuclei, and phagocytosis was visible. There was no hæmosiderin in these areas.

The pulp cells were closely packed cells with large open nuclei, and many were stuffed with granules and with material which had undergone phagocytosis. Eosinophile cells were present in small numbers.

In a few areas there was congestion of the sinuses with red cells, and in these areas there was a considerable amount of hæmosiderin which tended to be paravascular in distribution. The vessels were conspicuous by reason of the amount of perivascular connective tissue. The smaller vessels showed an increase of nuclei in the medial layers. This appearance is seen in normal spleens.

The microscopic appearances of the spleen showed considerable activity of the reticulo-endothelial tissue; the general histological appearance is otherwise normal, and is in no way suggestive of either acholuric jaundice or Addisonian anæmia (see Figure V).

Comment.

The diagnosis of anæmia in infancy and early childhood must be made with full recognition of the active growth period in which it occurs, and the necessity for constant marrow expansion which allows little opportunity for the development of a hæmopoietic reserve.

The loss of balance between erythropoiesis and erythroclases may occur very rapidly under a variety of conditions, and the resultant anæmia may be of very different significance from the similar blood picture in an adult.

In this case there were six outstanding facts.

1. The absence of any septic focus, hæmolytic poison, or other discoverable disease.
2. The persistently low total red and reticulocyte count.
3. Evidence of accumulation of the products of red cell destruction in serum and urine.
4. Absence of fragility and spherocytosis of the red cells.
5. Failure of response to liver therapy.
6. Normality of white blood cells.

The first point established the case as one of disorder of the hæmopoietic system alone. The next two suggested that it was of the nature of an erythronoclastic anæmia. The fourth, together with the family history, eliminated the diagnosis of familial acholuric jaundice; the fifth, the suggestion of an infantile type of Addisonian anæmia; while the normality of the white cells distinguished the condition from the subchronic hæmolytic anæmia described by von Jaksch.

A diagnosis of acquired erythronoclastic (hæmolytic) anæmia was therefore made.

Treatment.—In young children the prompt use of citrated blood in suitable amounts is necessary in the treatment of any form of anæmia; it was freely used throughout the course of this case. The early therapeutic measures were directed to filling the stores of building stones for hæmoglobin and red cells in the hope that the child's hæmopoietic system might again so balance itself that no further treatment would be necessary. This actually did happen in a similar but milder case under observation at the same time. The patient's inability to do this was clearly shown in the serial blood counts (see accompanying table), and by the relapse in the presence of infection.

Removal of the spleen was decided upon as an attempt to balance red cell formation and destruction by removing a depot where destruction is known to occur.

The physiological response of increase of red cells and platelets was obtained and maintained, and it was interest-

TABLE I.

Date.	Red Cells in Millions per Cubic Millimetre.	Hæmoglobin Value.	Colour Index.	White Cells per Cubic Millimetre.	Neutrophile Cells.	Lymphocytes.	Myelocytes.	Eosinophile Cells.	Reticulocytes.	Platelets per Cubic Millimetre.	Immature Cells.	Other Tests.
April—												
6 ..	1·55	30	0·97	7,200	40	50	2	6	None		2 normoblasts	Group 3.
							Transfusion					
9 ..	2·48	48	0·98	14,000	68	22	5	4	1·6%		1	Van den Bergh test, indirect positive reaction. Urobilin "++" in urine.
13 ..	2·23	44	1·0	6,900	49	43	4	3	None		1	Fragility, 0·36 to 0·33% saline solution. Van den Bergh test, indirect positive reaction.
15 ..	2·04	38	0·9	6,100	47	49	2	2	None			Fasting gastric juice, absence of free hydrochloric acid.
							Transfusion					
19 ..	2·52	52	1·02	4,000	54	30	4	12	Scanty		1 normoblast	Fragility, 0·36 to 0·33% of saline solution.
21 ..	2·57								None			
22 ..	2·67								0·83%			
23 ..	2·60	42	0·89	6,900	67	24	2	5	0·5%			Wassermann test gave no reaction.
25 ..	2·20								Under 0·5%			
27 ..	2·05	38	0·9	5,200	49	38	9	4	None			
							Transfusion					
29 ..	2·44	45	1·0	6,000	32	56	10	2	None	75,800		Faint urobilin in urine.
May—												
3 ..	2·02	38	0·9	5,700	61	33	4	2	None			
							Sternal trephine					
6 ..	2·00	36	0·9	4,600	52	32	8	4	0·8%			
							Transfusion					
13 ..	2·18	37	0·9	5,800	44	48	6	2	2·4%			
17 ..	2·28	36	0·8	7,500	60	28	2	8	2·3%			
24 ..	2·12	35	0·8	4,600	68	27	3	2	3·4%			
30 ..	2·00	40	1·0	8,000	60	32	8	0	3·3%			
June—												
6 ..	2·44	35	0·8	7,300	45	46	3	6	5·0%		2 normoblasts	Fragility, 0·45 to 0·36% saline solution. Urobilin "++" in urine.
15 ..	2·72	45	0·8	8,400	55	34	9	1	3·2%		1 myelocyte	
21 ..	3·10	55	0·9	7,300	53	42	4	1	3·0%		1 myelocyte	
30 ..	2·74	51	0·9	8,700	44	44	7	5	2·6%			Van den Bergh test, no reaction.
July—												
6 ..	1·91	34	0·9	6,400	46	44	6	4	1·0%			Fragility, 0·45 to 0·36% saline solution.
9 ..	1·65	25	0·78	9,100	41	51	4	4	None			Urobilin, "++" in urine.
							Transfusion					
12 ..	2·33	36	0·79	8,900	41	44	13	2	Under 1·0%	46,400		
16 ..	1·82	27	0·79	9,000	58	30	10	2	3·7%	104,700		
19 ..	1·60	27	0·84	6,700					11·0%	57,600		
							Transfusions: 19·7, 22·97					
23 ..	3·70	66	0·89	5,600	62	25	6	7	3·8%	92,500		
26 ..	4·30	73	0·84	7,100	62	28	4	6	2·3%	146,300		
							Splenectomy					
28 ..	4·60	65	0·78	7,800	76	16	6	2	Under 1·0%	306,000		
30 ..	4·40	67	0·79	14,000	68	11	10	11	Under 1·0%	440,000		Trace urobilin in urine
August—					5				Under 1·0%			
5 ..	4·34	73	0·85	14,000	55	23	10	12	Under 1·0%	571,000		Fragility, 0·45 to 0·36% saline solution.
13 ..	4·78	74	0·8	8,400	62	30	6	2	Under 1·0%	610,000		Van den Bergh test, no reaction. Urine clear.
19 ..	5·05	82	0·82	14,900	42	38	4	12	Under 0·5%	606,000		

ing to see that the mean average diameter of the red cells decreased slightly.

Summary.

A case of acquired erythronoclastic (hæmolytic) anæmia is described. Diagnosis and treatment are discussed.

Acknowledgements.

This patient was under the care of Dr. Lorimer Dods, to whom I am indebted for the privilege of making this report. Acknowledgements are due also to Dr. P. L. Hipsley, the surgeon, and to Dr. K. Winning, who gave the many blood transfusions.

THE FIBRO-FATTY LIVER.[1]

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WHEN I gave the title of this paper I expected to be describing a pathological lesion differing from the common portal cirrhosis. However, on further study of the case to be reported and of the pathology of the liver generally, I found my title quite incorrect; and so I shall correct myself now, and state my subject as a study of the ætiology and histogenesis of cirrhosis of the liver.

My interest in this subject was stimulated during a *post mortem* examination, in which a subject showed a particularly large liver, which was grossly cirrhotic. I shall give the case in detail.

Case History.

J.W., a male, aged forty-seven years, was admitted to the Adelaide Hospital on May 26, 1937, at 11 p.m. He was extremely ill on admission and a good history was difficult to obtain. He became ill the day before admission to hospital, with weakness in the arms and legs and a pain in the back of the neck. He was troubled by a severe pain in the left side, especially when he coughed. He had apparently always been well up to the present illness, except for some shortness of breath when running upstairs. He had had a course of injections in the arm, apparently intravenous, in 1929, for an unknown disease. He also stated that he had consumed considerable amounts of alcoholic beverages during the last few years.

Examination revealed a middle-aged male lying flat in bed in considerable respiratory distress. The skin had a yellowish tint in artificial light. The temperature was 36·59° C. (97·8° F.), the pulse rate was 108, and the respiration rate 48 per minute. The systolic blood pressure was 105, and the diastolic blood pressure was 60 millimetres of mercury. Examination of the heart revealed no abnormality other than an occasional dropped beat. A friction rub was discovered in the chest in the left mid-axillary line over the seventh and eighth ribs, and posteriorly there were dulness to percussion, diminished breath sounds and prolonged expiration over the eighth, ninth and tenth ribs on the left side. Examination of the abdomen revealed that the liver was palpable 7·5 centimetres (three inches) below the costal margin on the right side. The right knee jerk was sluggish and the left knee jerk was absent; both ankle jerks were absent, and the plantar response was flexor. There was slight œdema of the ankles.

Urine examination revealed that the specific gravity was 1020, and that albumin and a trace of acetone were present, but no sugar.

A diagnosis of left basal pneumonia was made, and palliative treatment was instituted. The patient died at 9.20 a.m. the day after admission to hospital.

A *post mortem* examination was carried out on May 27, 1937. This revealed an obese male body, with brown hair streaked with grey, of normal male distribution. The skin and conjunctivæ were stained a yellowish colour. *Rigor mortis* was not present; *post mortem* lividity was pronounced in all dependent parts. There was some œdema of the ankles.

When the abdomen was opened the liver was seen to extend one hand's breadth below the right costal margin and three fingers' breaths below the left costal margin, over the spleen.

On the chest being opened the lungs did not collapse. No abnormality was detected in the heart. The whole lower lobe and half the upper lobe of the left lung were solid, and the surface of the lung over this area was dark red and smooth. The cut surface was greyish-red in colour and homogeneous. The bronchi were congested. The right lung was partially collapsed, not crepitant, but no solid areas were detected.

The surface of the liver was grossly nodular, the nodules varying in size and the largest being about the size of a pea. The colour was a yellowish-grey. The capsule was thickened and what looked like streaks of pus were seen between the nodules. The liver was cut moderately easily, and the cut surface contained irregular lobules. The weight of the liver was 3,810 grammes (127 ounces).

The spleen was enlarged, of a prune juice colour, with many yellowish spots on its surface. The cut surface was dark in colour and considerably firmer than normal. The weight was 270 grammes (nine ounces).

No abnormality was detected in the kidneys, apart from the fact that they dripped blood on section. Nothing abnormal was found in the remaining organs.

Histologically the alveoli of the left lower lobe of the lung were filled with polymorphonuclear cells, some large septal cells and red blood cells. The alveolar capillaries were engorged with blood. All the blood vessels and the glomeruli of the kidneys were intensely engorged. Proliferation of the cells of Bowman's capsule was seen in a few glomeruli.

The cells of the liver were in the great majority filled with fat, the nucleus being displaced to an eccentric position. The cells were here and there arranged in columns, but in many places this arrangement was lost. The liver cells were divided into small and large lobules by fibrous tissue. Some of these lobules contained central veins and the cells had a radial arrangement, while other lobules contained simply a nest of cells, with no definite arrangement and no central vein. In the lobules the cells approximating the fibrous tissue appeared to be actively dividing, but no mitoses were seen. The fibrous tissue contained many bile ducts in its substance. With a methylene blue and phyloxine stain no hyaline granulations in the cytoplasm of the liver cells could be detected.

The pathological diagnosis was left lobar pneumonia involving the whole of the lower lobe and the lower half of the upper lobe; cirrhosis of the liver with fatty infiltration; fibrosis of the spleen (by a misfortune no section of the spleen was made); congestion of the kidneys.

Discussion.

The case brings up the old discussion of the ætiology of cirrhosis. That the disease was cirrhosis of the liver is quite clear from the histological picture, although the term cirrhosis seems to have no uniformity of definition in the literature. Some pathologists make a distinction between perilobular fibrosis and cirrhosis, using the latter term when there is definite alteration of the normal histological pattern of the liver. From the experimental viewpoint this seems justified, as many animals, such as rabbits and pigs, are very prone to perilobular fibrosis with age, and such lesions have been interpreted by experimenters as cirrhosis due to their own pet ætiological agent. Whether such a distinction is justified in human pathology is an open question, but it seems to me that it would lead to more uniformity in the literature.

In reviewing the work that has been done on the ætiology of cirrhosis, it seems that alcohol has been virtually disproved as a sole cause of cirrhosis. No work of any value has ever been able to produce cirrhosis with alcohol. However, alcohol does produce lesions in the liver. Fahr, in a small series of rabbits, working

[1] Read at the fifth session of the Australasian Medical Congress (British Medical Association), August, 1937.

with pure alcohol, found that the rabbits all had fatty infiltration of the liver after the administration of the alcohol. Freidenwald, in a series of one hundred and twenty rabbits, giving half the group absolute alcohol and the other half whisky, found fatty infiltration of the liver in both groups, this also being present in the heart and the kidneys; with the discontinuance of the alcohol this fatty infiltration disappeared. These findings have been confirmed by Bischoff and by Schafir, both these workers using whisky.

Alcohol in association with other agents can cause cirrhosis, but so can many other things. In association with phosphorus, alcohol very quickly produces cirrhosis in the rabbit. Mallory has produced the same lesions in the liver with phosphorus alone. Hurst and Hurst were able to produce cirrhosis with manganese and phenylhydrazine, and with manganese and *Bacillus coli*. It would be interesting in this connexion to study carefully the livers of patients receiving prolonged treatment with phenylhydrazine for polycythæmia or of manganese for staphylococcal infections.

Other agents which may produce cirrhosis in the laboratory animal are (i) chloroform with alcohol or bacteria, (ii) bacteria alone, (iii) tar alone, or with bacteria or alcohol. From the actual standpoint of the ætiology of human cirrhosis, bacteria or their toxins appear to be the most important factors, for in any community we are constantly bombarded by pathogenic organisms of varying virulence.

One point which may prove of importance is the observation made by Moon of the greater frequency of cirrhosis in the goitre belts of the United States of America. Whether this is only coincidental or of ætiological significance is as yet unknown. Wills has shown that goitre belts in India coincide with greater bacterial infection of the water supplies, and that the goitre incidence decreases with purer water supplies. This could serve as an explanation for the higher incidence of cirrhosis in these communities.

With regard to the histogenesis of cirrhosis, there seems to be little added to the general knowledge since Mallory's work in 1911. On one point pathologists are not in perfect agreement, and that is the question whether the hyaline granules in the liver cells are really evidence of an active lesion. Moon states that in ten cases of cirrhosis, those in which he could not find these granules were not diagnosed clinically, while in those in which they were present, all the patients had died with symptoms referable to their cirrhosis. If this is so, pathologists have a valuable means at their disposal of placing a cirrhosis in the active or latent class. I should be very interested to hear the opinion of pathologists on this subject.

As I stated at the beginning of this paper, my interest in this subject was stimulated by finding a liver of so-called atrophic cirrhosis, weighing one hundred and twenty-seven ounces. It was my wish to explain this lesion. Had the liver become very fatty, perilobular fibrosis then developing? The histological picture showed that we were dealing not with simple perilobular fibrosis, but with true cirrhosis, with destruction of normal liver architecture. With the methylene blue phyloxine stain no hyaline granulation could be detected, and if Moon's observations are correct, the cirrhosis was not an active one. Therefore it is reasonable for us to assume that the cirrhosis was of long standing and apparently clinically symptomless.

From the patient a history was obtained of heavy alcohol consumption over the past few years. Why not, then, endeavour to explain this liver condition on the basis of experimental findings? The patient contracted his cirrhosis from some toxic agent, and, this toxic agent later disappearing, the cirrhosis became quiescent. The patient consumed a considerable amount of alcohol over several years, and this caused in his liver a fatty infiltration of the cells.

My explanation of this lesion is probably open to many pitfalls to the pathologist, but it seems to me better than to term it an alcoholic cirrhosis, as is so often done when the patient gives the slightest history of the use of alcohol. It would be interesting to follow back the clinical histories of patients with simple fatty livers *post mortem*, to see if they had been heavy consumers of alcohol.

It has always seemed peculiar to me that alcohol should be so emphasized as an ætiological agent for cirrhosis. However, pathologists generally seem to be dropping this theory. In all the experimental work that has been done on cirrhosis, the sole agent that has universally given a true productive hepatitis has been bacteria of one type or another. We know that the normal liver is continually being bombarded by bacteria from the intestines, and to get liver tissue uncontaminated by bacteria is practically impossible. The ingenious experiment of Rous is interesting in this connexion. To get sterile Kupffer cells he injected fine iron filings into animals, and with strong magnets drew these cells containing the iron filings to an easily accessible spot. Even with this technique he was rarely able to get sterile collections of Kupffer cells. If, then, we have an organ continually bombarded by bacteria, why should these not produce a chronic hepatitis in some individuals, as common inhabitants of the throat produce chronic tonsillitis in one person and not in another? Undoubtedly other factors need to be considered; Mallory has produced cirrhosis with phosphorus, but he tries to prove it is a contaminant of spirituous liquors, unsuccessfully I might add. Manganese, chloroform and arsenic have all produced lesions leading to cirrhosis, and probably in some cases are the ætiological agents in human cirrhosis; nevertheless, it does seem evident that the *modus operandi* in the majority of cases must be the bacterium, and we as pathologists would be better advised to search out an organism from our cases of cirrhosis than to stifle our investigations in alcohol.

Summary.

A case of a large fatty cirrhotic liver, weighing one hundred and twenty-seven ounces, is reported.

The hypotheses are propounded that the cirrhosis was due to some unknown ætiological agent which had become inactive, and that the fatty infiltration was due to alcohol excess.

A short review of the experimental work on cirrhosis is given.

Bacteria are suggested as the main ætiological factors in cirrhosis.

MYOSITIS OSSIFICANS DUE TO INJURY OF THE BRACHIALIS ANTICUS UNASSOCIATED WITH FRACTURE.

By H. S. NEWLAND, M.S., F.R.C.S.,

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THE following is an account of an unusual injury.

On February 23, 1937, E.R., aged twenty-two years, was leading a bull with his right hand grasping the ring through its nose when the animal attacked him. He retained a firm hold on the ring until the bull pushed him through a fence.

His right elbow was injured in the struggle, pain being followed by swelling and bruising. The stiffness which was at first present decreased for a time under the ministrations of a bone setter.

When the patient consulted me on June 3, 1937, at the request of Dr. D. E. Drever, he complained that his ability to flex and extend the elbow joint was very limited. He felt no pain whatever.

On examination the contour of the bones of the elbow joint was found to be normal except for the presence of a hard fixed mass in front lying beneath the tendon of the biceps. The joint could be flexed to a right-angle only, and could be extended to about 135°. Each movement terminated abruptly in the resistance characteristic of bone. A radiograph taken by Dr. Drever revealed the condition seen in the accompanying figure.

The tearing of the *brachialis anticus* at its origin during the struggle led to bleeding and to the liberation of osteoblasts, hence the development of the *myositis ossificans*.

RECURRENT PASSIVE COLLAPSE OF THE LUNG.[1]

By H. STUBBE, M.B., B.S. (Melbourne), F.R.F.P.S. (Glasgow),
Honorary Surgeon, Fremantle Hospital, Western Australia.

A MALE patient, twenty-one years of age, when first seen in January, 1931, complained of a cough which had lasted for ten weeks. In 1916 he had undergone operation for tuberculous glands of the neck; this operation resulted in the dropping of his right shoulder and the protrusion of his right shoulder blade.

Examination revealed an apparently healthy young man with an old scar on the right side of his neck and wasting of the right trapezius muscle. Clinical examination disclosed dulness on percussion and absence of breath sounds at the base of the right lung. X ray examination, carried out by Dr. Gibson, revealed a dense opacity in this area, sloping downwards from the region of the seventh dorsal vertebra to the costo-phrenic angle. Some mottling of the lower edge of the body of the vertebra suggested caries. In view of the patient's history of tuberculous glands, the suspicious appearance of the vertebra, and a positive reaction to the Mantoux test, a diagnosis of tuberculous abscess of vertebral origin was made. The spinal accessory nerve paralysis suggested that there might also be involvement of the phrenic nerve, with elevation of the diaphragm. The patient was therefore put to bed in a plaster jacket for six months. When this was removed, and X ray photographs showed a normal right lung, he was congratulated and sent to a cattle station in the north-west of the State.

In May, 1936, the patient reappeared, with the same type of cough, clinical signs and X ray picture as in 1931. But on further X ray examination it was noted by Dr. Gibson that the irregularity of the seventh thoracic vertebra was an unimportant Schmorl's nodule, and that the trachea, mediastinum and heart were pulled towards the right (see Figure I). A revised diagnosis of passive collapse of the lower lobe of the right lung was made, and the patient was given an expectorant mixture. Two weeks later his cough, clinical signs and the shadow shown in the X ray photograph had disappeared. He refused bronchoscopic examination. The recurrence of this condition suggested the probability of a polypus in the bronchus acting as a ball-valve and permitting the escape of expired air, but preventing its entrance in inspiration.

ELUSIVE CALCULI.[1]

By H. STUBBE, M.B., B.S. (Melbourne), F.R.F.P.S. (Glasgow),
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A FEMALE patient, aged fifty-five years, was first seen in June, 1936, when she complained of extreme weakness, dyspnœa on exertion, and pain in the right loin during the preceding three weeks. Her family history was eventful. Her father, a brother and a sister had died of "heart disease"; her mother had died of "apoplexy", and another brother of "Bright's disease"; one brother who was then living was "paralysed after a stroke". The patient herself had had one full-term stillborn child and eight miscarriages. Four years prior to being seen by me she had undergone hysterectomy for "bleeding tumours".

Examination revealed a very pale, thin and anxious woman. Her remaining teeth were badly infected with pyorrhœa. The spleen was palpable four fingers' breadth below the costal margin. A firm, tender mass occupied the whole of the right hypochondrium and loin. The urine was found to contain a trace of albumin; its urea concentration was over 2%. Blood examination by Dr. Michaels revealed a secondary anæmia, with a leucocyte count of 11,000 per cubic millimetre. It was noted that many of the white cells were immature, which suggested the possibility of an aleuchæmic leuchæmia. Both the blood and the cerebro-spinal fluid gave a negative response to the Wassermann test. Cystoscopy revealed a normal bladder and ureteric orifices. The right ureter could not be catheterized, but the left was easily entered. Indigo-carmine was freely excreted from the left side within three minutes, but prolonged observation failed to discover any efflux from the right ureter. Pyelography carried out by Dr. Gibson gave interesting results. A preliminary film showed that four calculi were present in the right loin area, two of them lying two inches below the others, about the level of the umbilicus (see Figure I). The next film showed no "Uroselectan" appearing on the right side, but within five minutes an irregular, elliptical shadow, without any normal arrangement of the calyces, appeared on the left side. Stereoscopic films taken later, with the patient erect, showed that the lower pair of calculi had been joined by one of the upper ones, increasing the number in the lower group to three (see Figure II). A lateral view displayed the remaining upper calculus lying well in front of the vertebral bodies.

It was concluded that the function of an abnormal left kidney was good, that the right kidney was probably the site of calculous pyonephrosis, and that a single gall-stone was present. Operation was performed and a large pyonephrotic sac was removed intact, with about two inches of dilated ureter. Later, however, when the specimen was examined, it was found to contain no calculi at all. Since operation the patient has put on weight and considers herself cured, but she still has a large mass in the right hypochondrium. Cholecystography shows no gall-bladder shadow, but the four calculi *in statu quo*—one above and three below. Final diagnosis of the condition is hydrops of the gall-bladder containing four calculi, one of them probably being impacted in the cystic duct.

[1] The patient described herein was shown at a meeting of the Western Australian Branch of the British Medical Association on October 22, 1936.

CUSHING'S SYNDROME.

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AND
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IN 1932 Harvey Cushing outlined a syndrome which he showed to be associated with basophile adenomata of the pituitary gland. He postulated the following features as characteristic of his cases: rapidly acquired adiposity of face, neck and trunk, an alteration in normal hirsuties, a plethoric appearance of the skin with purplish *lineæ atrophicæ*, a tendency to kyphosis, a sexual dystrophy, vascular hypertension, a tendency to erythræmia, variable backaches, abdominal pains, fatigability and weakness. Other less consistently recorded features are acrocyanosis, purpura-like ecchymoses, aching pains in the eyes, dimness of vision, suggestive papillœdema, retinal exudate and hæmorrhage, œdema of the lower extremities, albuminuria, and polymorphonuclear leucocytosis.

Although prevfously a similar syndrome had been described under the name of the polyglandular syndrome in 1912 (Cushing,[(1)] Glynn[(2)]) and in 1926 (Parkes Weber[(3)]), attention had been mainly focused on the adrenal glands. In the last six years other observers have reported examples of this syndrome associated with other lesions, for example carcinoma of the thymus,[(4)] carcinoma and

adenoma of the adrenal cortex,[5] chromophobe adenoma of the pituitary,[6] and arrhenoblastoma of the ovary, and adenomata in both the pituitary and the adrenal[7] glands have been recorded as associated with Cushing's syndrome.

In contradistinction to the clarity of the clinical picture, the pathology of the syndrome remains relatively obscure. It has usually been assumed by workers that the most obvious glandular lesion in their particular cases has been the primary cause of the dystrophy. The above citations, however, suggest some causal factor common to all cases. Crooke[8] has recently examined the pituitary gland in cases of Cushing's syndrome associated with basophile adenoma of the pituitary gland, with tumour or hyperplasia of the adrenal cortex, and with tumour of the thymus. The only condition common to all was a peculiar hyaline change in the basophile cells of the gland, and this he interprets not as a cell degeneration, but as a probable altered physiological state. On the other hand, he examined the hypophysis in a large number of other conditions not associated with this syndrome, and found the hyaline change in very few glands, and then only in slight degree.

Basophile adenomata of the pituitary occur independently of this syndrome. Brauchli, Costello and Susman have individually examined the hypophyses of a great many (1,387 in all) subjects dead of disease of non-pituitary origin and have found adenomata of basophile type in 2·5%, in 4% and in 3·1% respectively.

Case History.

The following case now recorded illustrates many of the features of Cushing's syndrome. On May 29, 1937, H.H., a barman, aged twenty-four years, came under observation at St. George District Hospital. He complained of increase in weight, of headache, of breathlessness, of swelling of the feet, of watering of the eyes, and of inability to execute his duties.

FIGURE I.
The patient, anterior view.

As a youth his health was good and he was a keen athlete. In 1931, at the age of eighteen years, his mother first noticed that his face appeared to be gradually growing fatter; in 1932 his abdomen began to swell; in 1934 redness of the face became conspicuous, and in 1935 striæ appeared on the abdomen. There were occasional hæmorrhages from the rectum, always of bright blood, and up to a cupful in amount. During the last two years he became somnolent and was often overcome by lethargy in the forenoon. During the last twelve months headache, chiefly frontal, swelling of the ankles and feet, periodical polyuria and polydipsia occurred and became gradually more pronounced. His body weight had increased by twenty-two pounds in six weeks, and three months before admission to hospital it reached 84·37 kilograms (13 stone 4 pounds). There was no alteration of libido. There was nothing of significance in his previous or family history.

Clinical examination showed an obese male, looking older than his stated age (Figures I and II). He was mentally alert and cheerful. The face was big, red and plethoric. The obesity, localized to the face, neck and trunk, contrasted strongly with his relatively thin arms

FIGURE II.
The patient, lateral view.

and legs. There was no hirsuties. The very tense and prominent abdomen was marked by numerous purplish striæ. The fat was not tender or painful. The external genitalia were normal. The pulse rate ranged between 90 and 120 per minute and the rhythm was regular. The systolic blood pressure was 190 and the diastolic pressure 115 millimetres of mercury. Retinoscopy revealed advanced bilateral retinitis, with scars, exudate and hæmorrhages more extensive on the left side. Pronounced albuminuria was present, but there was no glycosuria. Systematic examination yielded no other abnormal findings.

Microscopic examination of the urine revealed numerous hyaline casts and occasional granular casts.

The following investigations were carried out.

The results of the urea concentration test (Maclean) are shown in Table I.

TABLE I.

Time.	Percentage Urea.	Urine in Cubic Centimetres.
Before draught	1·95	142
1 hour after draught (15 grammes urea)	1·90	34
2 hours after draught	2·45	100
3 hours after draught	2·05	58

A blood count made on June 8, 1937, gave the following information. The erythrocytes numbered 5,650,000 and the leucocytes numbered 15,200 per cubic millimetre. The hæmoglobin value was 115% and the colour index was 1·0.

In a differential count, neutrophile cells made up 83% of the total leucocytes, basophile cells 1%, lymphocytes 14% and monocytes 2%.

The serum calcium was estimated at 10·6 milligrammes *per centum*. There was no reaction to the Casoni test.

The radiological reports were as follows. Examination of the skull revealed that the pituitary fossa was normal in size. No erosion of either the anterior or the posterior clinoid processes was found. A plain skiagram of the abdomen revealed that the upper pole of the left kidney was obscured and compressed by a soft tissue mass above it, which mass showed some evidence of calcification (see Figure III). Pyelography revealed that the left renal system showed evidence of compression; acute pain was experienced after the injection of 1·5 cubic centimetres of sodium iodide solution. The upper calyx system was somewhat distorted. There was some contraction of the renal pelvis (see Figure IV).

During his stay in hospital the patient's condition remained unaltered, with the exception that the œdema of the feet and ankles disappeared and he developed ecchymoses on the extremities, both spontaneously and as a result of slight traumata.

A blood count on July 6, 1937, revealed that the erythrocytes numbered 4,340,000 per cubic millimetre and thrombocytes 150,000 per cubic millimetre, and that the hæmoglobin value was 62%.

On July 8, 1937, a left suprarenal tumour was removed through a left oblique subcostal incision. Considerable difficulty was experienced in exposure of the tumour on account of the increased size of the liver and the abundance of intraperitoneal fat. The tumour itself was shelled out with ease. After operation the patient's condition was fairly satisfactory. His pulse rate, however, remained elevated and he died twelve hours later in a condition resembling profound shock.

At autopsy the abdominal fulness was found to be due almost entirely to fat, not in the panniculus, which was nowhere of greater thickness than 1·9 centimetres (three-quarters of an inch), but in the omentum and mesentery. The liver was large and fatty and weighed 2,280 grammes (four and three-quarter pounds). Left ventricular hypertrophy was observed in the heart.

The right suprarenal gland was atrophic and difficult to isolate in the surrounding fat, which was present in excess amount, and the gland was represented by a narrow strip of tissue, 0·3 centimetre (one-eighth of an inch) thick and 3·1 centimetres (one and a quarter inches) long. The left suprarenal tumour, lobulated and roughly ovoid in shape, measuring 12·5 by 7·5 centimetres (five by three inches) and weighing 360 grammes (twelve ounces), was of pale yellowish colour and of firm consistency (see Figure V). On section the lobulation was more evident, and areas of calcification and necrosis could be defined.

We are indebted to Dr. G. F. S. Davies for the following histological report.

> The suprarenal tumour is a carcinoma. It has a sinusoidal structure, but the columns of cells between the sinusoids are wide and the cells are closely packed together. The cells are irregularly polygonal in shape and their cytoplasm is abundant and deeply eosinophilic. Nuclei show a moderate degree of irregularity in size and shape, and considerable hyperchromatism. Running through the tumour are thick strands of fibrous tissue which contain large blood vessels, some of which are thrombosed. There is a well-defined fibrous capsule on the surface of the tumour in the part sectioned. One section contains an irregular area of necrosis in which the original structure may still be recognized, but here selective staining of nuclei is lost.
>
> The right suprarenal gland: the cortex is narrow and division into the usual three layers is not distinct. A few cell clumps corresponding to the *zona glomerulosa* are dark staining, but the remainder have clear foamy cytoplasm.
>
> There is no *zona fasciculata* and no pigmented cells are seen. A few clumps of displaced cortical cells are seen in the medulla. The medulla varies in amount in different parts. In one section there is more medulla than cortex, and the cells are dark staining and moderately well preserved, and in another only a few clumps of medullary cells are present.
>
> Pituitary: No adenoma is present. In the *pars intermedia* there are numerous large alveoli, which contain eosinophilic and slightly foamy material. The *pars posterior* shows no obvious abnormality.
>
> The pancreas shows *post mortem* degeneration.

Subsequently, serial sections of the whole pituitary gland were prepared, and examination of them failed to reveal an adenoma. For this examination we are indebted to Dr. Latham.

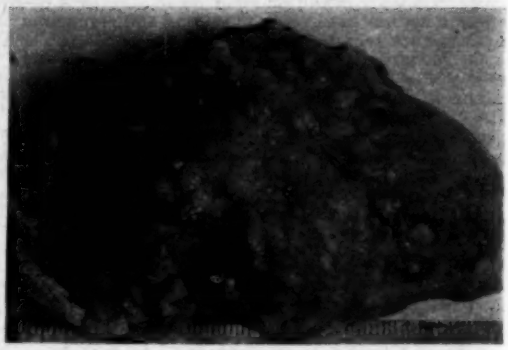

FIGURE V.
The tumour (scale in inches).

Discussion.

Diagnosis.

The site of the lesion being, as before mentioned, so variable, the main problem is its location rather than the recognition of the syndrome, the features of which are usually so characteristic. Hence the endocrine organs require systematic examination. The skull should be examined by means of X rays for possible abnormality of the pituitary fossa. If the cause is a basophilic adenoma, changes discoverable by radiography will seldom be found, since such tumours are of minute size (the diameters being 3·0 and 4·0 millimetres and 2·5 millimetres in Cases VI and VII respectively of Cushing's series). An attempt should be made to demonstrate clinically and radiologically the presence of a tumour of the suprarenal glands. A plain skiagram will reveal these tumours, if they are of any size, because of their density and of calcification, which is sometimes present, as illustrated in our case and in that of Graham Lescher. Simple cortical hyperplasia, the more commonly occurring condition, would not be demonstrated in this way.

Pyelography is invaluable. In a characteristic case the kidney is displaced downwards, the upper pole is compressed, and the upper calyces are distorted. The ureter, however, does not emerge from the anterior surface of the kidney, as is the case in the unascended and usually unrotated organ. The mobility and the type of subject in which it commonly occurs should assist in differentiating the merely ptosed kidney. There is a radiological technique[20] whereby it is claimed that the suprarenals can be outlined. This is useful in determining not only the presence of a tumour, but also the normality of the opposite gland. The chest should be radiologically examined for secondary nodules and for enlargement of the thymus. The finding of increased amounts of follicular sex hormone in the urine, especially in males and in females past the menopause, would be of diagnostic significance, as would be the loss of normal ovarian sensation on vaginal examination.[21a]

Treatment.

If the investigations as outlined are inconclusive, two lines of procedure may be followed: (i) laparotomy or

(ii) irradiation of the pituitary. Some workers, experienced in these conditions, recommend exploration of the adrenals by laparotomy, and in some cases have gone further and removed the larger of two apparently normal glands. We, however, doubt the wisdom of employing a major operation for diagnostic purposes, seeing that in these patients surgical intervention is fraught with more than ordinary danger. Bland and Goldstein[11] state:

> Patients with pituitary basophilia cannot withstand operative measures and are poor risks for any type of surgical procedure, as shown by the fact that nine patients succumbed after operation of one type or another. Five patients were operated upon for suspected adrenal neoplasm and they all died following partial or total adrenalectomy (Freyberg, Lescher, Fuller, Reichmann and Bauer). It is of absorbing interest to note that in only one of these patients was an adrenal neoplasm found at operation. Later, at autopsy, a minute basophilic adenoma was discovered in the pituitary gland (Lescher). One patient, described by Moehlig, died following thyroidectomy. Of two patients operated upon for the removal of pituitary tumour, one succumbed, while the other made a good recovery with cessation of the symptoms (Lisser). One patient, who had improved somewhat as a result of pituitary irradiation, died subsequently after an appendicectomy (Wright). One case terminated fatally from infection following direct pyelocystoscopy.

Therefore it is advisable first to subject the pituitary to intensive irradiation and carefully to observe any results, such as return of menstruation in the female, restoration of more normal bodily form and remission of other prominent signs—hirsuties, hypertension *et cetera*. In occasional cases this improvement has with persistence gone on to apparent cure. The minute size of a basophile adenoma militates against its surgical removal. Should adequate irradiation fail to effect clinical improvement, laparotomy is to be considered.

Operative Technique.

For the removal of a suprarenal tumour three routes are available, the transpleural, the lumbar and the transperitoneal routes, and for the latter various incisions have been employed. Broster and Vines[12] prefer the transthoracic route. Other authorities, however, draw attention to the possibility of thoracic complications, and to the fact that exploration of the opposite adrenal is impossible by this method. A further fact for consideration is that the blood supply of the suprarenal glands, which is a rich one, is distributed mainly to their antero-medial aspects. This and the position of the tumour relative to the ribs should be remembered in deciding for or against the lumbar route. For adequacy of exposure, safety of operation and completeness of removal the transperitoneal route appears to be the best. The right side is the more difficult to deal with on account of the relation of the gland to the *vena cava*, into which its central vein drains. If the tumour is large it may encroach upon the *vena cava*. On the left side the gland is partly covered by the pancreas, and the splenic vessels cross its lower pole, so that injury to these structures must be carefully avoided. Its central vein is more accessible and drains into the renal vein, which makes control of the blood supply easier. After exposure of the tumour these veins should be secured; thus manipulation will not produce embolic metastases or a flooding of the circulation with adrenaline. In the present case adequate exposure was obtained through a generous oblique subcostal incision.

Despite early control of the venous connexions, these operations are commonly attended by considerable shock. Cecil[13] states that the literature shows that 39% of the patients have died from shock soon after removal of these tumours. Of those who recovered, it has been reported that 66% had severe shock.

Lisser quotes a case reported by Holl as the first instance of recognition and successful removal of an adrenal cortical tumour in an adult male with cure of the internal secretory disturbances. He was able to find only five cases in the literature of adrenal cortical tumours in adult males causing endocrine disturbances.

If for any reason operation cannot be carried out, deep X ray therapy may be used. Apert and Dubost have reported favourable results from irradiation of the tumour area, and Crile and his associates report remarkable improvement in a girl aged seventeen years after bilateral denervation of the adrenals and a partial adrenalectomy of one gland.

The prognosis in Cushing's syndrome is poor, and the course of the disease rarely exceeds a few years. This would be expected in the cortical tumours, as many of them are malignant. Recurrence and metastases have caused death in the majority of recorded cases, even after apparently successful removal. In basophile adenoma it is little better, although one patient observed by Cushing survived for more than twenty years.

Summary.

An account of Cushing's syndrome and an outline of treatment are presented and a case is recorded.

There is some diversity of opinion as to the pathogenesis of the condition and, when a lesion elsewhere has been assigned as the cause of the syndrome, doubt has been expressed as to the condition of the pituitary gland. Unfortunately this has rarely been thoroughly investigated. Therefore we have been at pains to have the pituitary microscopically examined in serial section. An adenoma was not found; and this, together with a full autopsy, indicates that the cortical adrenal tumour alone was responsible, whatever its action may have been on the pituitary function.

There are other features of interest in this case: firstly, the rarity of the condition in adult males; secondly, absence of alteration in libido (the patient was engaged to be married); thirdly, the length of the history (six years), and the absence of metastases. Late metastasis is characteristic of cortical adrenal carcinoma.

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A SELF-INFLICTED SMALL PERFORATING WOUND IN THE SKULL CAUSING AN ABSCESS IN THE VENTRICLES.

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SERIOUS head injuries due to self-mutilation by mental patients are not always associated with dramatic display and immediate and obvious signs. From a medico-legal point of view they are interesting, as at times the clinical and physical signs closely resemble those caused by disease. The following lines illustrate these points.

Clinical History.

(John McGeorge.)

R.F., aged forty-eight years, had a long criminal history and was eventually declared an habitual criminal. He served two years of his sentence and then developed hallucinations, believing that he heard his uncle's voice saying, "Shed thy blood". He gashed his arms and attempted to batter his head against the wall. He was certified insane, and was sent to the Criminal Mental Hospital at Parramatta.

He exhibited a gradual mental deterioration with expansive delusions, and was often manneristic.

After eight years in hospital he drew attention one day to a small papule in the mid-line just above the bridge of the nose. This had a minute opening in it, and the skin around was congested. He claimed that he could introduce a piece of wire through this into his brain, and stated that his object was to allow the escape of insects from his head. A few days later he complained that he did not feel well. His temperature was then 37° C. (98·6° F.), his pulse rate 94 and respiration rate 20 per minute. The following day the temperature was 38·3° C. (101° F.), the pulse rate was 104 and respiration rate 24. He complained of persistent pains in the neck and occiput, and head retraction was present. Lumbar puncture was performed and the cerebro-spinal fluid revealed an increase in polymorphonuclear cells, a decrease in glucose, and was turbid. No organisms were isolated. His condition improved for a time, but ten days later he had a convulsive seizure with coma and stertorous respiration. Twitching of the left arm and hand occurred with adductor spasm of the extremities. On stimulation of the plantar surfaces a movement of withdrawal was elicited on the right side, with a doubtful Babinski reflex on the left side. The knee jerk on the right side was exaggerated, on the left it was clonic in type. Meningismus and Kernig's sign were present, but the patient's temperature was only slightly raised until two days before death, when it became subnormal. He died twenty-five days after the onset of the first symptoms.

At autopsy all organs were healthy. There was a considerable quantity of purulent fluid in the cranial cavity and the pia was markedly congested. A small funnel-shaped opening extended from the outside into the cranium. The internal opening was about the size of a pin-point. It was situated in the mid-line in the frontal region 1·5 centimetres above the glabella. There was a definite area of congestion with radiating hæmorrhagic striæ around this. The dura was adherent to the inner surface of the skull at the site of the puncture. The brain was removed intact and forwarded for pathological examination.

The formalin fixed brain was coronally sectioned through the anterior part of the anterior commissure, and again further back through the Rolandic sulcus parallel with the first after Wertham's method.

The Course of Infection.

(Gilbert Phillips.)

The specimen was well fixed and included both cerebral hemispheres from the tips of the frontal lobes as far back as a coronal section made at the level of the tips of the temporal poles. A rectangular slab of tissue (10 by 3 by 10 millimetres) had been excised from the prefrontal area on the left side at the junction of the medial with the supero-lateral surface.

The third ventricle was full of purulent material, which was also found to cover the floor of the bodies of the lateral ventricles, particularly on the right side. Bilateral asymmetrical internal hydrocephalus was present, the right lateral ventricle being distended more than twice as much as the left. The cerebral surface displayed no gross naked-eye abnormality. On section an abscess cylindrical in shape and varying from 1·0 to 1·5 centimetres in diameter was found to extend forward from the floor of the right lateral ventricle in the region of the foramen of Monro. This abscess, full of purulent material, reached to within one or two millimetres of the tip of the right frontal pole, its median edge being only one or two millimetres deep to the medial surface of the right cerebral hemisphere at a depth of three centimetres from the right supero-medial convexity.

Pathological Notes.

(Oliver Latham.)

Pieces of tissue were removed from the neighbourhood of the skull perforation, the ventricle and abscesses, from various distant parts of the brain stem and cortex. They were stained by hematoxylin and eosin, alizarin red, Nissl's methylene blue and by von Braunmuhl's stain for senile plaques, which also reveals young fibrils and glial excess. Naturally the usual inflammatory reactions were found in the neighbourhood of the abscesses and skull perforation, chiefly new vascular formations and hypertrophy of the old with glial hypertrophy where colliquative necrosis was not extreme. The most interesting phenomenon, however, was the relative confinement of the neurone damage to the actual lesions. The thickened pia, subpial felting and gliosis might well have been an accompaniment of the man's mental condition. However, there were widespread, if slight, evidences of pial reaction to the wound in the nature of mononuclear cells, pus cells and extravasated blood cells in many regions in the pia arachnoid. The neurones were remarkably well preserved throughout the brain, the Betz cells especially escaping. A smear of the pus revealed pus cells, cocci and bacteria.

A similar case was described to me by the late Dr. Chishom Ross. Some forty years ago a mental patient had applied an iron nail two and a half inches long to his forehead, and had then bumped his head against the lavatory wall frequently. The wound was dressed, but its nature was not recognized. Eventually he died, and at *post mortem* examination the head of the nail, which had perforated the skull and brain, was found beneath the scab on his forehead.

In THE MEDICAL JOURNAL OF AUSTRALIA, June 16, 1934, I briefly alluded to two cases in which the skull was perforated from within. In the first I was called to perform a *post mortem* examination on a middle-aged woman with clinical signs of acute *encephalitis lethargica*. I noticed a slight sore on the inner side of her left eyebrow which had a dressing on it. A probe could be passed right into this wound and through the skull into the brain. On opening the skull a sarcoma or neuroblastoma of the left frontal lobe was found which had perforated pia, dura and skull just above the cribriform plate, apparently affecting the skin. Penfield described a similar case in which a neuroblastoma of the occipital lobe had perforated the occipital bone to form a tumour in the neck.

Summary.

A case has been presented wherein a small piece of wire was applied so effectually to the forehead that it

eventually punctured both skull and brain, causing intraventricular abscesses. A similar case in which a nail was used has been alluded to, and comparisons have been made with like perforations in the skull produced from within by brain tumours.

OSSIFICATION CENTRE FOR A SACRALIZED LUMBAR VERTEBRA.

By H. FLECKER, M.B., Ch.M., Dip.Rad. (Sydney), F.R.C.S., *Cairns, Queensland.*

RADIOLOGISTS are by now quite familiar with the appearance of the so-called sacralized lumbar vertebra, when on one or both sides the fifth lumbar vertebra develops an ala similar to the alæ present on the first three sacral vertebræ and developed from the separate centres for the lateral masses of the sacrum. Such sacralized lumbar vertebræ are seen very frequently and either remain distinct or fuse with the sacrum upon one or both sides. When fusion does not take place the sacralized process is accused of impinging in certain circumstances against the sacrum and causing considerable discomfort. In any case such anatomical irregularities are said to cause various low backaches. However, many such abnormalities, which have not produced any symptoms whatever, have been discovered quite by accident.

The subject of this demonstration is a girl, aged three years and seven months, in whom this abnormality was discovered quite by chance, as she did not complain of any symptoms referable to the spine.

Upon the left side the fifth lumbar vertebra is perfectly normal, but upon the right there is a solid ossification centre with an epiphyseal plate joining it to the lower margin of the transverse process and neighbouring portion of the neural arch. Its medial margin forms the outer border of a foramen between the fifth lumbar vertebra and the sacrum. The lower border does not as yet reach the sacral ala, but may in the course of development either reach it and form a sutural articulation or may possibly fuse with it, as frequently happens. (See the accompanying figure.)

That this ossification centre is in series with the lateral masses of the sacrum is evident from this view, where the suture between the sacral vertebral bodies and lateral masses is not yet fused.

The absence of the first coccygeal segment is of special interest, not only because it is rarely absent after birth, especially in females, but it indicates a tendency towards cephalad shifting of the vertebral structures, such as has resulted in the formation of the sacralized lumbar vertebra.

Reviews.

RHEUMATIC DISEASES.

DR. F. J. POYNTON AND DR. B. SCHLESINGER have published a second edition of their important work on rheumatic diseases.[1] Apart from its contents of clinical interest, this book is of great value for the information it gives us concerning the progress of the anti-rheumatic organizations in Great Britain and in the United States of America. From this information it is apparent that in this respect Australia is dropping a long way behind the other English-speaking communities as the years go by. It was in August, 1912, that Dr. F. J. Poynton suggested that special convalescent homes would be the means of averting much of the chronic heart disease of rheumatic origin. In 1915 Dr. W. St. Lawrence inaugurated an experimental clinic at Saint Luke's Hospital, New York, from which have spread the extensive schemes of America, England and other countries. In 1918 Dr. Poynton, from his ward at the Hospital for Sick Children, Great Ormond Street, London, organized a scheme of special convalescent homes and supervisory and treatment centres. In 1923 Dr. Leonard Findlay set up an experimental clinic at the Royal Hospital for Sick Children, Glasgow, on the lines of St. Lawrence in New York. In 1931, when the first edition of the book under review was published, five special rheumatic convalescent homes had been in existence for some years in America, and from 1919 special recovery hospitals had been opened in England. From the efforts of the Invalid Children's Aid Association, the first, the Edgar Lee Home, was opened in 1919, and a second and larger home, Kurandaf, was opened in 1924 in Sussex and transferred in 1927 to West Wickam, Kent, with accommodation for 80 beds and with specially designed open-air wards. In 1921 Baskerville, with 90 beds, was set aside for the special educational treatment of children recovering from chorea or rheumatic heart disease as part of the comprehensive scheme organized in Birmingham. Ordinary convalescent homes were partially converted, and in many places certain wards were reserved under the supervision of doctors with special knowledge and interest in the subject. The Metropolitan Asylums Board of the London County Council had 152 convalescent beds for rheumatic children in 1931 and then announced its intention to have another 350. We learn in the new edition of the book that in 1937 in America organizations for prevention, investigation, treatment, supervision and study of rheumatic infections are widespread and very active. Big centres exist at New York, Boston, Philadelphia, Chicago, St. Louis and other cities throughout the country. In England the problem soon became too urgent and extensive to be dealt with entirely by individual or charitable organizations. The London County Council has at its disposal close on 800 beds for convalescent rheumatic children. It has not yet been decided how many recovery hospitals will be required, but hospitals of 80 or 90 beds are regarded as the most economical in size. Partially converted convalescent homes and maintenance by voluntary efforts are embarrassing features which have not been a great success, though they have filled the gap. Many of these places are still in operation, though they are considered "unsuitable for the treatment of children with rheumatic heart disease". Facilities for medical research and clinical study of the disease and educational and vocational training are features of the network of clinics and hospitals.

We cannot escape the feeling that two definite groups of diseases are discussed in the book without adequate demarcation. The group of diseases perhaps unsuitably designated "juvenile rheumatism", and inclusive of rheumatic heart disease and chorea of rheumatic origin, could with advantage be made completely separate from the other important group of so-called rheumatic diseases of a deforming and crippling arthritic nature, which lead to so much inefficiency and loss of work in older subjects. The problems of investigation, prevention and treatment of these two diseases may require separate solution, and the whole field is too great for individual research workers and organizers. Until it is proved that the two groups of rheumatic diseases have a common origin, it would be well to keep them further apart, even to the point of making them the subjects of separate books in the "Recent Advances" series.

The usefulness of the book is greatly enhanced by the references that have been brought up to date. By means of these references the student of rheumatic infections is helped considerably to trace the whole vast literature of this fascinating subject, and in addition he is given definite indications in the text to aid in the decision as to which of the references are likely to be worth following up for the special object in view.

The specificity of streptococci in the ætiology of the rheumatic diseases is no longer stressed by the authors, and an interesting account is given of the recent work of Schlesinger, Signy and Amies on the virus as an ætiological factor. Insufficiency of vitamin *B* and vitamin *C* is discussed as accessory and predisposing to rheumatism, and damp surroundings, though doubtful factors in the

[1] "Recent Advances in the Study of Rheumatism", by F. J. Poynton, M.D., F.R.C.P., and B. Schlesinger, M.A., M.D., F.R.C.P.; Second Edition; 1937. London: J. and A. Churchill Limited. Large crown 8vo, pp. 390, with 51 illustrations. Price: 15s. net.

incidence, are regarded as liable to provoke symptoms in latent cases. The pathological changes in rheumatism are considered in three stages. The first is the stage of fibrinoid swelling in connective tissues and muscles, with focal necrosis and a tendency to hæmorrhages. The stage of absorption and attempted repair follows and is succeeded by the stage of scar formation. The authors think it probable that the pathology is non-specific and that the Aschoff nodule cannot be regarded as the essential lesion. The behaviour of rheumatic diseases in the connective tissue skeleton has been elucidated further, and the opinion is expressed that the subcutaneous nodule appears to be one of the main links between acute and chronic rheumatic diseases. Involvement of the intestine and peritoneum is discussed, and in this feature another point is added to the resemblance between the clinical behaviour of rheumatic and tuberculous diseases.

No one should use "Nirvanol" therapy for chorea or for subacute carditis or active rheumatism without studying the excellent account of the subject given by these authors, who, with necessary safeguards, range themselves on the side of those clinicians who claim to be able to get good results with the drug mentioned.

The book by Fortescue Fox and Van Breemen published in 1934 has influenced the present authors in revising the first edition to the extent of adoption in part of their classification of chronic rheumatism. Dr. Howitt's chapter on physiotherapy has been revised, and an interesting new feature has appeared in the chapter on surgical treatment of chronic rheumatism from the pen of Mr. Eric I. Lloyd.

We have no hesitation in recommending this new edition to our readers, and we urge them to use it freely and fully. It is well printed and well indexed and is very informative.

MODERN PSYCHOLOGY IN PRACTICE.

IN "Modern Psychology in Practice" Dr. W. Lindesay Neustatter has written a book primarily for the practitioner and medical student.[1] He is deserving of congratulation for writing a volume which will be helpful. As Dr. Gillespie states in his foreword: "It is written in readable and even at times in homely language and is based mainly on observed facts."

The subject matter falls naturally into five sections. The first deals with psychopathology. Whilst the author has admittedly Freudian leanings, he shows a pleasing clarity of expression and an appreciation of the views of Adler, Jung and Stekel.

Section II concerns disorders of children and will be particularly useful to the general practitioner. Anxious and nervy children, temper tantrums, lying and other behaviour traits, stammering, incontinence, and mental deficiency receive brief but fairly adequate mention. Two particularly interesting chapters deal with play therapy and vocational guidance. The second is written by Mr. Jack Jennings, of the National Institute of Industrial Psychology. He draws attention to the remarkable results achieved in evaluating a child's capabilities towards employment. The inadequacy of Australian facilities in this respect is only too obvious. The diffusion of this type of knowledge should help to stimulate our authorities into further action on this most necessary and vital part of social service.

The neuroses and psychoses in adults are described in the third section. Particular attention is paid to cases which present difficulties in treatment, for example the prognosis of melancholics, the suicide problem, how to deal with relatives.

Section IV is devoted to treatment. The author stresses the need for an amoral outlook by the physician. He may state the case, but the patient must decide the ethics. "He is, so to speak, the patient's counsel, and not the judge, let alone the prosecuting K.C."

The last section deals with psychology and general medicine. Dr. Neustatter rightly stresses the need for considering the psychological factors in asthma, rheumatism, chorea and rheumatoid arthritis.

In a small book of this description it is easy to find deficiencies. No mention is made of allergy as a factor in enuresis; no description is given of the singing method in the treatment of stammerers; *anorexia nervosa* and pituitary disorders are not differentiated; the use of sedatives in melancholia receives scant attention. Where space is so limited he would be indeed a carping critic who would withhold praise on account of the unsaid. The volume is worth a place in the library of the busy practitioner.

[1] "Modern Psychology in Practice", by W. L. Neustatter, B.Sc., M.B., B.S., M.R.C.P., with a foreword by R. D. Gillespie, M.D., F.R.C.P., D.P.M.; 1937. London: J. and A. Churchill Limited. Large crown 8vo, pp. 214. Price: 10s. 6d. net.

PHYSIOLOGY AND THE PRACTICE OF MEDICINE.

THE volume entitled "The Physiological Basis of Medical Practice", by Best and Taylor, is a very comprehensive contribution to the subject.[1] The authors state in the preface that they "have endeavoured to write a book which will serve to link the laboratory and clinic and which will therefore promote continuity of physiological teaching throughout the pre-clinical and clinical years of an undergraduate course". In this purpose they have succeeded admirably. The whole range of physiology is covered in a work which includes 1,519 pages of text and 100 pages of references.

The authors have not hesitated to step at times into the realms of pathology in their endeavour to link the laboratory and clinic. This is illustrated in the chapter dealing with the pathological physiology of kidney diseases, in which are discussed the classification of kidney disease, renal insufficiency, some of the more useful tests of renal function, albuminuria, casts, polyuria, oliguria and uræmia. The chapter on hæmoglobin serves as an illustration of the more academic side of the book, in which porphyrins, metallo-porphyrins, heme, cytochrome and hæmocyanin are most interestingly considered. On the other hand, the chapter on the anæmias is almost entirely clinical in its outlook.

Throughout the whole book is this intimate association of the academic and clinical viewpoints. The volume is uniformly good and is very suitable for teachers of physiology and clinical medicine, and for the more advanced classes of students of physiology.

THE DIETETIC CONTROL OF DIABETES.

THE fifth edition of "The Diabetic A B C", by R. D. Lawrence,[2] contains a few additions which are concerned with the use of protamine insulin preparations and which are stated in the preface to be "what the patient needs to know about the practical uses of the new compounds". This is stated very briefly, and it is disappointing to find that no alterations have been made in the partition of the diets which exemplify the line system, in order to render it more applicable to the new insulin.

In all other ways the book remains the same, and is an excellent manual for the exposition of a simple and satisfactory scheme of dietetic control of this disease. It would seem, however, that Lawrence's statements on protamine insulin are if anything too concise, and that he could have included with advantage some of the details which are to be found in the latest edition of his other book, "The Diabetic Life".

[1] "The Physiological Basis of Medical Practice", by C. H. Best, M.A., M.D., D.Sc., F.R.S., F.R.C.P., and N. B. Taylor, M.D., F.R.S., F.R.C.S., F.R.C.P., M.R.C.S., L.R.C.P.; 1937. London: Baillière, Tindall and Cox. Medium 8vo, pp. 1705, with illustrations. Price: 45s. net.

[2] "The Diabetic A B C: A Practical Book for Patients and Nurses", by R. D. Lawrence, M.A., M.D., F.R.C.P.; Fifth Edition; 1937. London: H. K. Lewis and Company Limited. Demy 8vo, pp. 71. Price: 3s. 6d. net.

The Medical Journal of Australia

SATURDAY, FEBRUARY 26, 1938.

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POST-GRADUATE EDUCATION IN NEW SOUTH WALES.

THE history of post-graduate education in medicine in Australia has been traced in this journal on several occasions. When the Melbourne Permanent Post-Graduate Committee was formed many years ago the members of the profession recognized the value of the "refresher" courses that were held every year, and senior and junior practitioners availed themselves regularly of the opportunities of study provided for them. The Branches of the British Medical Association in other States of the Commonwealth soon followed the example of the Victorian Branch, and committees were founded in all the States. When comment has been made in these pages on progress in the organization of post-graduate medical teaching in important centres, the history of the undertaking in question has been reviewed. This is necessary in order that a proper perspective may be obtained and the significance of the change may be realized. An organization, complete and satisfactory in every respect, can rarely if ever be brought into being without a preliminary period of slow and perhaps painful development. Even if there appears to be no reason why full function should not at once be attained, a latent period will generally occur—the several parts of an organization, like those of a machine, have to be "run in".

In November, 1935, we drew attention to the formation under the auspices of the University of Sydney of the New South Wales Post-Graduate Committee in Medicine. This body took the place of the New South Wales Permanent Post-Graduate Committee. It will be remembered that the Permanent Post-Graduate Committee was formed at the instance of the New South Wales Branch of the British Medical Association. The New South Wales Permanent Post-Graduate Committee did a great deal for practising members of the medical profession in arranging refresher courses, week-end courses in country centres, week-end courses in special subjects at metropolitan hospitals, special courses in ophthalmology, cardiology, laboratory methods, and so forth, as well as courses for the primary examination for the Fellowship of the Royal College of Surgeons of England. When the New South Wales Post-Graduate Committee in Medicine came into being, the personnel of the committee remained the same as under the old regime, except that the Chancellor and Vice-Chancellor of the University became *ex officio* members. The new committee has thus the prestige and authority of the University behind it. Emphasis was laid on these aspects in this journal a little more than two years ago. At that time we drew attention to the urgent necessity for the establishment of a post-graduate hospital where approved students could, by a kind of apprenticeship system and under the guidance of experienced teachers, not only bring up to date their knowledge of the rudiments of medicine and surgery, but undertake study in special branches of medical science. This need will shortly be satisfied.

For very many years the Prince Henry Hospital, Little Bay, previously known as the Coast Hospital, has been of the greatest value to the members of the community, particularly those in poor circumstances and those suffering from infectious diseases. The Government of New South Wales, which controlled the Prince Henry Hospital, thought that the institution would be more effectively administered by a board than by departmental officials. At the same time it was anxious to provide a hospital where post-graduate training might be carried out. It therefore drew up and passed the *Prince Henry Hospital Act*, 1936. According to the act, the hospital is controlled by fifteen directors. Of these, one is a member of the Hospitals Commission of New South Wales, who is not a member of the medical profession; two are appointed on the nomination of the Senate of the University of Sydney; one is appointed on the nomination of the Council of the Royal Australasian College of Surgeons; one is appointed on the nomination of the Council of the Association of Physicians of Australasia; one is appointed on the nomination of the New South Wales Post-Graduate Committee in Medicine of the University of Sydney; and eight, who must not be members of the medical profession, are appointed on the nomination of the Minister. It is also directed that the chairman of the board must not be a member of the medical profession. Clause 25 of the act provides for the establishment of post-graduate teaching in the following terms:

> The Board shall make such provision as may be necessary or desirable to enable post-graduate teaching and research work in medicine to be carried out in the hospital by or under the authority of the University of Sydney.

In another section it is stated that it shall be a primary duty of the board to make available at all times not less than three hundred beds for the treatment of infectious diseases. The New South Wales Post-Graduate Committee in Medicine realized that the Prince Henry Hospital would not make an ideal institution for post-graduate teaching—the institution is a long way from the city, the number of out-patients is small, and it may be necessary at any time to make extensive provision for patients with infectious disease. Expert advice was sought as to whether it would be economically advisable to convert two recently erected large blocks into wards for the treatment of infectious disease, since the wards that were formerly used for patients with infectious diseases were obsolete and quite unsuited to the requirements of a modern hospital. It was thought that the money saved in this fashion might be used to build in a more suitable position a central post-graduate hospital, which would be an annexe to Prince Henry Hospital and under the control of the same board of management. The advice of experts was against the adoption of this course. In the circumstances the New South Wales Post-Graduate Committee in Medicine decided to make the fullest possible use of the present facilities at Prince Henry Hospital. With the approval of the board, the Senate of the University of Sydney has appointed a senior physician and a senior surgeon who will take charge of the medical and surgical units and who will supervise post-graduate tuition. Provision is also being made for the appointment of a director of pathology.

Everyone will agree that the Prince Henry Hospital is not ideally constituted or situated to serve as a post-graduate hospital; everyone, however, will be forced to admit to a feeling of satisfaction that the Government of New South Wales has realized the necessity for post-graduate study in medicine. To have refused to accept Prince Henry Hospital as a post-graduate teaching hospital would have been foolish in the extreme. The Board of the Prince Henry Hospital and the New South Wales Post-Graduate Committee in Medicine will, we trust, have the support and cooperation of all unprejudiced medical practitioners. Both bodies, faced, as they are bound to be, with many difficulties, will welcome frank criticism from those who have constructive suggestions to offer. The medical profession in New South Wales and beyond its boundaries has the opportunity of helping to create an institution which will be useful to them and to their successors in the years to come.

Current Comment.

THE URINARY EXCRETION OF IRON.

For many years progress in the pharmacological knowledge of iron remained almost completely in abeyance. In recent times, however, the hiatus in our understanding of iron, particularly in regard to its absorption and elimination, is being rapidly contracted. Even now there are major divergencies of opinion regarding many phases of iron metabolism. Adelaide P. Barer and W. M. Fowler remind us that there is a considerable difference of opinion as to the quantity of iron excreted in the urine.[1] They point out that some of the discrepancies may be due to variations in the sensitivity or the technique of the method of estimation used. But this does not explain the great variation in the results obtained by some observers, who found no iron in the urine, and those of Riecker, who found 19·79 milligrammes in twenty-four hours in a case of pernicious anæmia. In an endeavour to explain the discrepancy, and as no comparison had been made between the output iron in the urine of men and of women, Barer and Fowler undertook an investigation of one hundred men and one hundred women. None was included whose urine contained blood, and the subjects were all allowed to continue their usual life as far as diet and medication were concerned. Patients, however, who received supplementary iron or excessive fluid were excluded. In women, analyses were undertaken during the intermenstrual period. The iron content of the diet was calculated from the tables of Dorothy S. Waller, and was taken as the approximate daily iron intake. Complete specimens of urine for three days were used as a basis for the study. Special precautions were taken to prevent iron contamination, and the urine was collected directly in glass containers. Every day creatinine excretion was determined to ensure that collection of the specimen was complete. Triplicate iron determinations were made on aliquots of the pooled three-day specimens after a modification of the method of F. Reis and H. H. Chakmakjian. Hæmoglobin was ascertained by the method of H. S. Newcomer, and a hæmocrit reading was made on all patients.

Barer and Fowler give extensive tables embodying the results of this study of urinary iron excretion. The ages of the patients varied between eighteen and seventy-eight years; but it was observed that there was no correlation between the ages of the patients and the iron excreted in the urine, nor between the creatinine excretion and the urinary iron loss. It was seen that some of the patients excreting more urine also eliminated slightly greater quantities of iron in the urine. But, as this was noted in only a small percentage of cases, it was not deemed to be of significance. No definite correlation between urinary volume and iron excretion, as had been suggested by G. E. Farrar and S. M. Goldhamer, was detected.

The iron intake in the diet of these subjects varied between 2·46 and 21·82 milligrammes daily, and no relationship was noticed between the urinary iron excretion and the iron intake within those limits. These results accorded with those of C. M. Coons, who had found that the quantity of urinary iron was not related to the urinary volume, nor was it proportional to the iron content of the diet. Barer and Fowler state that, as additional proof that the urinary iron excretion for each person is constant, regardless of the iron intake, ten of the subjects were given every day three grammes of iron and ammonium citrate. Urinary iron excretion was not materially altered by such supplementary iron. In another group comprising ten subjects the urinary iron excretion was estimated on a second occasion after a lapse of 30 to 235 days. It was found that the greatest variation between the two determinations in any subject was 0·1 milligramme per day. Barer and Fowler consider that their results indicate that urinary iron excretion may vary from one case to another, but is fairly constant for each individual.

The hæmoglobin in these subjects varied from 1·590 to 19·740 grammes per 100 cubic centimetres of blood; but no correlation was ascertained between the hæmoglobin level and the amount of iron excreted in the urine. The range of urinary iron loss is greater in women than in men. It was found that the average urinary iron in women was 0·489 milligramme daily; in men it was 0·395 milligramme, the difference being 24%. Barer and Fowler found no reason for this divergence, and wisely refrained from hazarding any explanation. It was seen that, in fourteen patients suffering from idiopathic hypochromic anæmia, the average iron excretion was 0·720 milligramme daily, or 47% more than the average urinary loss for the 100 women and 63% more than the average for men and women together. Barer and Fowler state that any importance of the greater urinary loss of iron in women is doubtful; but the fact that it is even greater in idiopathic hypochromic anæmia may enhance its significance. Barer and Fowler, while considering it possible that it may sometimes be a contributing factor in some instances, do not believe that this is the actual cause of the condition.

The contribution of Barer and Fowler is of particular value as an addition to our knowledge of the pharmacology of iron. Many more investigations on greater numbers of subjects are necessary before finality on these matters is attained. The discrepancies between the results of Riecker and others are not yet explained.

[1] *The Journal of Laboratory and Clinical Medicine*, November, 1937.

Abstracts from Current Medical Literature.

PÆDIATRICS.

Erythema Marginatum (Rheumaticum).

C. BRUCE PERRY (*Archives of Disease in Childhood*, August, 1937) draws attention to the association of *erythema marginatum* with rheumatic infection, and considers that it appears to be a specific rheumatic manifestation. The eruption characteristically starts as a solid erythema, little, if at all, raised. It gradually spreads out, and as it does so the skin in the centre of the lesion returns to normal, thus forming the typical spreading marginate or annular eruption. Where the spreading circles of rash meet they coalesce, forming a larger ring. The usual sites of the occurrence of the rash are on the front of the abdomen and on the front and back of the thorax. It may develop on the limbs, but the author states he has never seen it on the face. The rash may, and usually does, appear at the onset of the acute attack or relapse of rheumatism, but it is frequently present when there are no other signs of active infection and when the sedimentation rate is normal. It tends to come and go, lasting from one or two days to months or years.

Vitamin B_1 Deficiency in Infancy.

M. R. PRICE (*Archives of Disease in Childhood*, August, 1937) reviews the subject of vitamin B_1 deficiency in infancy. The amount of this vitamin required by an infant has not been ascertained, but a child appears to require relatively more than an adult for each pound of body weight. Increase in weight would seem to need increase of vitamin B_1, and a diet of high caloric value requires this vitamin for its utilization. Vogt-Muller states that the more carbohydrate a diet contains, the greater the requirements of vitamin B_1. Recent literature in England and America shows clearly how large a measure of agreement there is among various investigators concerning the prevalence of minor degrees of vitamin B_1 deficiency, especially in children. The symptomatology of such minor degrees of deficiency is varied and leads to widely differing clinical pictures, from the well-defined syndrome, such as pink disease, to the indefinite but frequently met with condition called debility or "failure to gain weight". The author draws attention to the actions which have been attributed to vitamin B_1 by several independent workers. These are briefly an increase in muscle tone, especially of the intestinal tract, a catalyst in carbohydrate metabolism, and an essential factor in the well-being of lymphoid tissues. From a consideration of these actions it is concluded that the result of adding vitamin B_1 to the diet of infants and children would be an increase in appetite and in assimilation and utilization of food, thus producing an increase in weight and height.

Hepatitis Epidemica.

PER SELANDER (*Kinderärztliche Praxis*, May and June, 1937) reviews in two papers the subject of sporadic and epidemic hepatitis (catarrhal jaundice). Predisposing factors are age (childhood and youth being more susceptible) and overcrowding (as in schools, prisons, barracks, camps and institutions). The cause is infection by a virus, probably by the droplet method. The infectivity, like that of other virus diseases, is not high, very short contact sufficing. Patients are certainly infective before the onset of jaundice. Laboratory workers have been infected from handling specimens of urine or serum. Care should be taken in the early stage, as cross-infection and infection of the nursing staff can occur. Patients are not strictly isolated. Abortive attacks confer immunity. The incubation period is two to four weeks. The onset and course of the disease are described in detail, and attention is drawn to the great variation in severity, ranging from mild and abortive cases, perhaps without jaundice, to severe long-continued attacks which may end fatally. Complications are rare. Secondary cholangitis and acute yellow atrophy occasionally occur. Acute nephritis is more common, coming and going with the jaundice. Cirrhosis of the liver may ensue, even when the initial acute stage has been apparently mild. These patients have recurring jaundice before the symptoms of cirrhosis set in. Diagnosis must be made particularly from Weil's spirochætal jaundice and, in adults, cholangitis. Cases may be mistaken for acute gastro-enteritis or appendicitis. There is no specific treatment.

Cerebral Tumours in Children.

RUBY O. STERN (*Archives of Disease in Childhood*, October, 1937) presents the results of the examination of one hundred and two cases of cerebral tumour in the Hospital for Sick Children, Great Ormond Street, during the period 1921 to 1935, occurring in children under the age of twelve years. A large proportion of the tumours, over one-half of the cases investigated, occurred during the first five years of life. This is at variance with most of the series hitherto published. The topographical distribution confirmed the observations of others, twice as many tumours being present below the tentorium as above it. It would appear, therefore, that in children the more primitive regions of the brain are affected by tumour rather than the regions more recently evolved. It might be supposed on similar grounds that the tumours present in children would consist of more primitive malignant cells than do those of adults. Actually this was not the case in the author's series, a slow-growing tumour with well-differentiated cells being as common as the more malignant types of tumour derived from nervous tissue. The hopelessness of these cases with benign tumours is due to their situation, which too often offers insuperable difficulties to a surgical approach. They kill by their site of election rather than by their nature or size. Contrary to what might be expected also, the majority of the benign growths occurred before the age of five years. No tumours of the pituitary fossa were encountered, and only one occurred in the supra-pituitary region. A histological study was made of fifty-five of the tumours. Two-thirds of these belonged to the glioma group. Well-differentiated gliomata were as common as the more malignant forms, but occurred in situations inaccessible to surgical treatment. Only one solitary tuberculoma was found. Tuberculomata were rare tumours, only six examples being recorded in the series.

Rickets Resistant to Vitamin D Therapy.

FULLER ALBRIGHT, ALLAN M. BUTLER AND ESTHER BLOOMBERG (*American Journal of Diseases of Children*, September, 1937) report the case of a boy of sixteen who had suffered from rickets since the age of one year in spite of treatment with vitamin *D*. Ordinary infantile rickets generally is considered a specific deficiency disease in which the quantity of specific agents, that is, vitamin *D* or ultra-violet rays, required for the prevention or cure of the disease depends particularly on the amount of calcium and phosphorus in the diet. Small amounts of the specific agents are usually sufficient to defend the average person on a normal diet from the disease. Steatorrhœa predisposes a child to rickets presumably because vitamin *D*, being fat-soluble, is lost in the fat of the stools. Renal insufficiency and disease of the liver predispose the patient to conditions resembling rickets, which, however, are not true rickets. Certain patients, however, in the absence of these predisposing factors, acquire true rickets when receiving an amount of the specific agent usually adequate for prophylaxis, and such patients fail to respond to doses that ordinarily effect a cure. There has been considerable doubt as to whether the decrease in the effectiveness of vitamin *D* in such patients is due to lack of absorption of the vitamin or to an intrinsic factor which has resulted in a resistance to the vitamin's action. In the case reported histological evidence demonstrating that the condition was true rickets and not some other condition resembling rickets is presented. Metabolic data are present to show that changes in the calcium and phosphorus metabolism were similar to those in ordinary infantile rickets, and evidence is also presented to show that the cause of the dis-

order was not a primary hyperparathyreoidism. The ristance to vitamin *D* therapy was not due to failure to absorb vitamin *D*, since crystalline vitamin *D* injected intravenously and radiation from an ultra-violet ray lamp were ineffective. The disturbance in this patient cannot be considered a deficiency disease like ordinary rickets, but rather a form of rickets due to an intrinsic resistance to the anti-rhachitic action of vitamin *D*. This resistance can, however, be broken down with massive doses of the vitamin.

ORTHOPÆDIC SURGERY.

Low Back Pain.

R. G. SPURLING, F. H. MAYFIELD AND J. B. ROGERS (*The Journal of the American Medical Association*, September 18, 1937) discuss the differential diagnosis between intraspinal and extraspinal causes for low back pain. The presence of objective neurological changes, such as sensory or motor loss about the buttock, impotence and possible disturbances of the sphincter, is in favour of an intraspinal lesion; herniation of a *nucleus pulposus*, neoplasm, inflammatory disease or hypertrophy of the *ligamentum flavum* may produce this clinical picture. Hyperplasia of the fibres of the *ligamentum flavum* is possible at any level, but in the authors' experience the lesion is limited to the ligaments connecting the fourth and fifth lumbar vertebræ and in many cases the lamina of the fourth lumbar vertebra was also greatly increased in thickness. The duration of symptoms in this group of patients varied from three months to two years, and pain low in the back was the predominant complaint. This pain arose suddenly while the patient was lifting a heavy object in the flexed position and was followed by a period of relief after a few days or a week, only to recur insidiously until the patient was incapacitated. It was sometimes unilateral, was aggravated by a sudden change of position, and radiated into one or both lower extremities. Diminution of sensibility, tenderness to pressure over the sciatic nerve, objective motor loss and diminution of the Achilles reflex were almost constant findings. When the neurological examination suggests an intraspinal lesion, examination of the cerebro-spinal fluid below the probable level of the lesion should reveal elevation of the protein content of fluid if obstruction is present, and the characteristic findings on fluoroscopic examination of the spine, after injection of two cubic centimetres of lipiodol, will give conclusive proof of the location of the lesion. The authors show serial photographs of such findings, but have been unable to demonstrate the hypertrophy of the fourth lumbar lamina in the radiograph. Treatment of this lesion consists in removal of the involved lamina and ligament. This has been done under local infiltration with procaine hydrochloride and has resulted in prompt relief of symptoms. Microscopic examination of the affected ligament has shown replacement of the normal yellow elastic tissue with white fibrous tissue in which calcareous deposits were present.

Treatment of Compound Fractures.

O. J. HERMAN (*The New England Journal of Medicine*, December 2, 1937) describes his treatment of compound fractures at the Boston City Hospital. While admitting that every compound fracture presents specific problems of what to do and when to do it, he claims that a fairly routine treatment up to the reduction and fixation point is an aid to expeditious work. The treatment of all fractures should begin at the scene of the accident with efficient first aid treatment, shock being minimized by hæmostasis, traction, fixation, early administration of hypnotics and proper transportation. On arrival of the patient at hospital the treatment varies according to the degree of shock, which, if severe, entails adequate treatment in the shock room. If shock is not severe, or when the patient has recovered from shock, he is taken to the X ray room and then to the operating floor, where, after anæsthetization, a most thorough cleansing of the wound and the surrounding operative field is done. Careful *débridement* of the wound, with special attention to the avoidance of unnecessary trauma to the healthy tissues, is done with instruments, which are frequently changed to avoid contaminating the fresh wound. After complete hæmostasis has been established the wound is gently and moderately irrigated with salt solution followed with a mild irrigation of alcohol, then with mercurochrome-acetone alcohol solution, extreme care being exercised to avoid the periosteum and bone as much as possible. After a complete change of instruments, gown, gloves and drapes, the bone ends are "gently curretted to remove all possible contamination". The wound is gently irrigated, but all bone chips that are not hopelessly devitalized or contaminated are preserved. Where necessary, internal fixation by plate, band suture or nail is unhesitatingly used to maintain adequate reduction and apposition, and these measures have not increased the incidence of sepsis. The author prefers primary closure of the wound without drainage by loose sutures, and does not hesitate to use lateral incisions to relieve tension. When internal fixation has been used, a plaster of Paris cast or moulded splint usually gives adequate external fixation, but most other cases require traction, probably in a suspended Thomas splint. The author treats all fractures of the pin-hole or puncture type by the same methods. Delayed union and non-union are minimized by careful attention to hygiene, physical therapy, the indirect stimulation of weight-bearing through the use of ambulatory splints, and, if these measures fail, by multiple drill holes or some form of bone graft.

Congenital Dislocation of the Hip.

A. R. SMITH (*Annals of Surgery*, July, 1937) discusses the symptoms of irreducible congenital dislocated hips, their incidence and the various age groups, and compares the results of treatment of cases which have resisted attempts to produce closed or open reduction. The three methods of treatment compared are the Shantz or low osteotomy, the Lorenz-Baeyer bifurcation and the shelving operation. The objective symptoms of limp, shortening, scoliosis and the subjective symptom of pain were by far the most reliable. In unilateral cases no patient complained of pain before the tenth year, and this was accompanied by fatigue as a symptom complex in the later age groups, but was frequently absent prior to the fifteenth year. The degree of pain and fatigue in the later age groups depends on the degree of arthritis present. The author points out that this symptom complex is necessarily absent in those patients who have never walked. In investigating the results of shelving, he limits his cases to those in which conservative methods or attempts at open reduction have failed, and presents the results of 28 operations on 25 patients, of whom 50% were in the age group of six to ten years. He finds that the operation is frequently followed by the necessity for secondary operations when the operation is done before the age of ten, whereas the reverse is the case after that age. Results in bilateral dislocations are much poorer than those in unilateral cases and several times a shelf was completely reabsorbed. The end result appears to be good when the anatomical results as shown radiographically are good; but all good functional results and all good anatomical results occurred in patients ten years old or older, and the author comes to the conclusion that the functional value of a shelf built before the tenth year is doubtful. He presents the results in five patients, all with bilateral dislocations, on whom eight bifurcation operations were performed. In only one case was pain present following the bifurcation, and he quotes Øwre's opinion that the bifurcation is best not done until the fifteenth year or later, because of the loss of the supporting angle in the younger patients. The number of hips requiring secondary surgical procedures following the bifurcation operation in these patients is also extremely high, being 33%; and in 66% of these cases the symptom complex of pain and fatigue was present after the bifurcation had been performed. The results of the Shantz or low osteotomy were equivocal in two cases.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at the Medical Society Hall, East Melbourne, on December 15, 1937, DR. J. NEWMAN MORRIS in the chair. The meeting was a special one, and a general invitation was extended to all legally qualified medical practitioners to attend.

Symposium on Present State of the Poliomyelitis Epidemic.

The Spread of the Disease.

DR. JOHN DALE, Medical Officer of Health of the City of Melbourne, said that in order to present briefly to those present a working hypothesis of the nature and spread of poliomyelitis he had to be dogmatic. In the first place it was necessary to realize that the virus in its ordinary or usual form was one of the most harmless of the common human parasites. There was evidence that it was widespread in all civilized communities. Most people, presumably, encountered the germ in infancy and early childhood and the majority of older persons were immune.

Dr. Dale said that in Great Britain, where epidemics had not occurred, there was a steady incidence of deaths widely scattered, mostly in young children, amounting to about four per million inhabitants. Even in Australia, where the tendency to epidemicity had in recent years been greater, the annual toll to date had been only about five per million. Relating these deaths to the flow of children into the community, he would say that of each 10,000 children born probably two would die of poliomyelitis, about four would be severely paralysed, and a number of others would be made ill, perhaps with some paralysis, but would not be recognized as suffering from poliomyelitis. In meeting another common parasite, the diphtheria bacillus, some 50 of the 10,000 would die, and some 1,500 would be recognizably and more or less severely ill. Dr. Dale pointed out that in the prevailing epidemic they had in Victoria a strain of poliomyelitis virus which was much more damaging and therefore more evident than the usual strain, although there was no evidence that the present strain was more infectious; the point was that the infections were more evident. The present virus was killing something like one in 400 instead of one in 4,000, causing severe paralysis in one in 200 instead of one in 2,000, and causing recognizable illness often with some paralysis in perhaps one in 50 or less of the susceptible children. He said that even in that epidemic it could be inferred that, when the first case of the disease was recognized in a new centre, there were probably from 10 to 40 people in the district already infected. In his own area, the City of Melbourne, they had details of 109 younger children exposed to infection from an older recognized case in the family. Among those seven definite cases had been diagnosed and eleven children had shown sickness regarded as "abortive" poliomyelitis. The incidence of seven cases in 109 presumably susceptible children might be regarded as high in view of what he had just said, but the factors of familial susceptibility and high degree of exposure had also to be considered. Curiously the series included four sets of twins, three homologous, in which one twin had the disease and one escaped. Obviously all those children had been infected and would be immunized.

Dr. Dale did not know whether the silent, sub-clinical or immunizing infections were as infectious as evident cases, but presumably they were capable of spreading the virus. He was inclined to accept the hypothesis, for which Dr. Scholes was mainly or entirely responsible, that, as in some other virus diseases such as measles, an infected person did not give off virus during the incubation period but then became relatively highly infectious for some days, perhaps a week, after which he was not likely to be actively or practically infectious, although he might, in special circumstances, be able to spread the infection. The hypothesis was supported by numerous instances in which persons showing slight symptoms about ten days after exposure had apparently been the infecting source of another case. If there were twenty or thirty silent cases which were infectious for every one evident case known to be infectious, it might be asked what was the use of isolating the one recognized case. In any case, the circumstances emphasized the general importance of measures designed to prevent unnecessary gatherings of children. Dr. Dale thought that such measures would, and probably had, slowed down the spread and prevented the sudden overwhelming of hospitals and other organizations, and surely that was a very important consideration.

The incubation period was not less than seven days and seemed to range between eight and twelve days. It was becoming generally accepted that the virus was strictly neurotropic, gaining access to the nervous system usually through the olfactory nerves and producing all the usual general, and later paralytic, symptoms in the course of its passage through the mid-brain down to the medulla and cord. The virus was spread from person to person presumably by the respiratory route in a manner somewhat similar to the spread of influenza.

Dr. Dale stated that the curve of the epidemic in Melbourne was a progression rather than a rapidly rising curve as in influenza. This was to be explained by the fact that only a small proportion of the population, mainly children, was susceptible, that the children were kept more or less isolated from parents and that the houses in Melbourne were mainly separate dwellings relatively well and widely spaced. In cities such as New York and Toronto, where families were largely packed in flats and apartments, the curve of the epidemic rose much more rapidly. Dr. Dale demonstrated slides which showed how in New Zealand the disease, breaking out in Dunedin in December, 1936, had, over the next six months, gradually spread northwards and covered both islands. It seemed probable that the virus had been brought from New Zealand to the suburb where the first cases had occurred in Melbourne.

Dr. Dale also showed slides and a film illustrating the creep of the virus over the metropolitan area. He showed that the foci of infection became established first in some areas and later in others, and how apparently some sparks blowing early to certain places had started fires which had gone out. At the end of six months, however, the majority of suburbs were heavily infected.

Dr. Dale said that, though the outbreak was the most extensive yet recorded in Australia, it was not yet comparable as regards deaths in Victoria to those occurring in New Zealand in 1924 and 1916. The New York epidemic of 1916, in which the deaths had reached a rate of 25 per 100,000, was many times more severe than the present epidemic to date. However, the virus was widespread in the country areas also, and, in view of the approach of late summer and autumn, the most dangerous period for poliomyelitis, it was impossible to say what might yet occur.

Dr. Dale showed other slides of age distribution which illustrated that 83% of the patients in the metropolitan area were under ten years of age, with the peak in the seventh year, though in the inner suburbs the peak year was the sixth; 96% of patients were under fourteen years of age; 60% were males. He said that the greater susceptibility of males was held to be a true sex difference. These features are illustrated in the accompanying figures.

The Diagnosis of Poliomyelitis.

DR. MOSTYN L. POWELL, Medical Officer to the Joint Government and Municipal Committee for Infantile Paralysis, Melbourne, after stating that he did not wish to repeat the subject matter of the paper read on July 29, 1937, and published in THE MEDICAL JOURNAL OF AUSTRALIA on September 11, 1937, said that he would attempt briefly to indicate certain symptoms and signs not usually seen in poliomyelitis but very commonly encountered in many of the acute and subacute febrile ailments of childhood. The diagnostic value of those findings from the negative side would thus become apparent and help medical practitioners to reach a decision when poliomyelitis as a diagnosis was under consideration. Before going further Dr. Powell emphasized the fact that there was no single sign or symptom which was always present or which was

pathognomonic of infantile paralysis; even neck stiffness was sometimes absent in the early stage and occasionally throughout the course in the mild cases. He made the same reservation in respect of the negative signs which were not perfect, but their presence in the history or in the examination should cause doubt to arise much in the way that a story of frequency and dysuria caused doubt in the diagnosis of acute appendicitis. Cough with or without coryza was a symptom which in Dr. Powell's experience had been exceedingly rare in poliomyelitis; even if it was not mentioned in the history he always asked for it and the differential diagnosis of poliomyelitis. He instanced as examples acute pain in the ear and in the side of the neck, a tender joint or ligament, chest pain and even limb pain if accompanied by tenderness. He stated that the back or limb pain of poliomyelitis might be quite severe at times and might even require morphine for its relief, but it appeared to be a dull type of pain and was unaccompanied by the grimacing and contortions evinced by the more severe acute pain and tenderness of the fibrositis, lumbago, osteomyelitis and cellulitis. When he had asked an intelligent boy who had had a lot of neck pain and

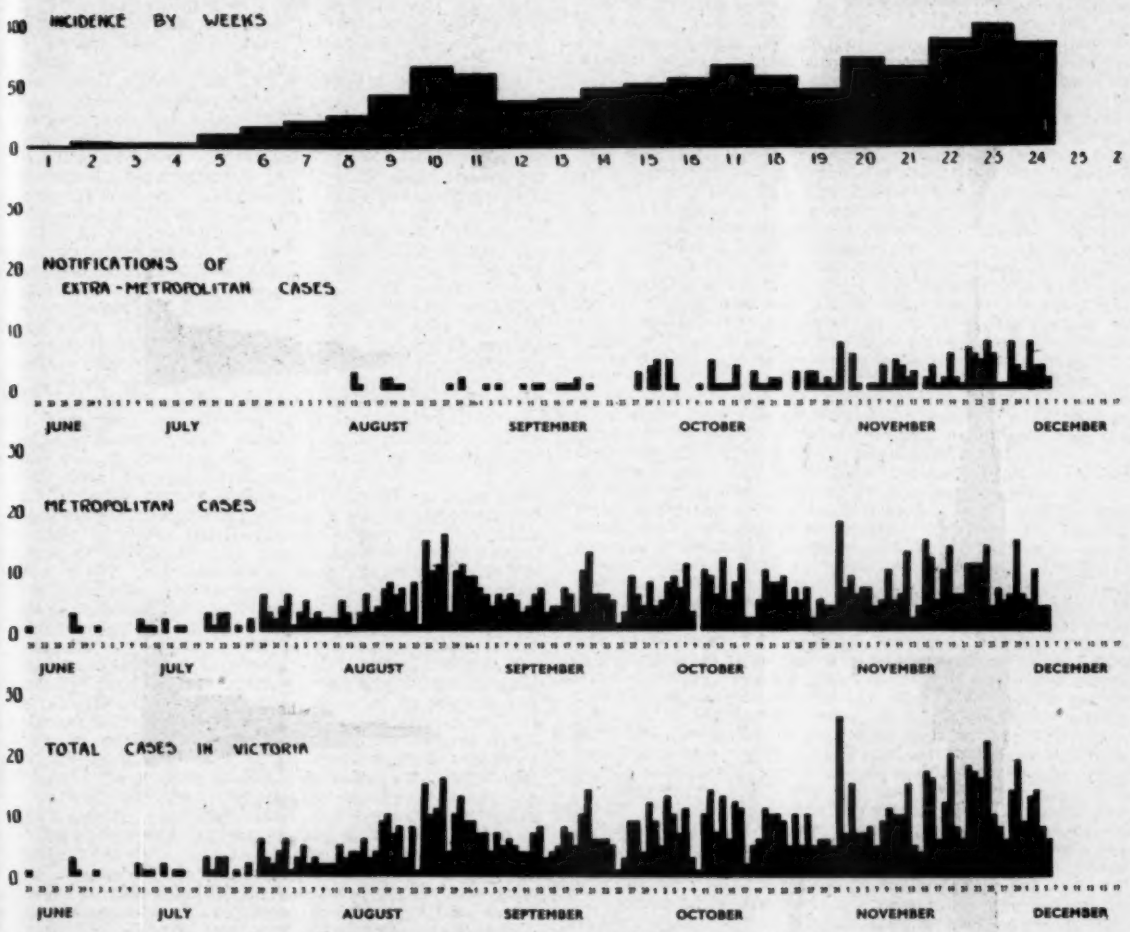

Figure showing incidence of poliomyelitis by weeks and days.

was on his guard at once if it proved to be present. To this statement two exceptions were mentioned. In poliomyelitis with pharyngeal involvement, when the inhalation of saliva or food caused cough the diagnosis was usually beyond doubt on the other evidence and the cough appeared as a late manifestation. The other exception was a coincidental "cold", though in his experience the coincidence was extraordinarily rare.

Diarrhœa as a symptom of common occurrence in the febrile phase of poliomyelitis was usually described in the text-books, but in the prevailing epidemic it was certainly not the case. If it appeared in the history it immediately turned Dr. Powell's thoughts from the intrathecal to the intraperitoneal cavity. Mild constipation or normal actions of the bowel had been the usual finding.

Severe acute unilateral pain and tenderness in any region had been found by Dr. Powell to be of special value in stiffness to describe the pain, the boy likened it to the shocks received on turning the handle of an old-fashioned magneto which was at his home. As the handle was turned faster and faster the shocks became more and more severe, running up the arms until he felt he could not stand it any more. That was the sensation that boy had experienced in the neck. A point of interest was that, irrespective of the degree of pain in the neck in poliomyelitis, the actual tenderness to palpation was very slight. Dr. Powell had found this fact of value in the differentiation of poliomyelitis from fibrositic, inflammatory or traumatic affections of the neck. At this stage he referred to the "head roll", which was a sign that had been useful in diagnosis. However great the degree of neck stiffness to anterior flexion had been in poliomyelitis, lateral rotation or rolling of the head had been amazingly free and usually had been possible without pain or stiffness

through the full range of movement. This sign had helped him to eliminate such conditions as acute myofibrositis of the neck groups of muscles which caused neck-stiffness, but the patients gave a characteristic grunt of pain when the head was rolled laterally in one direction or the other. When the neck stiffness was not due to poliomyelitis, localized tender spots either at the muscle insertions, in the muscle bellies or in the glands of the region had frequently been elicited and it had surprised Dr. Powell to find that there was strikingly little actual regional tenderness on palpation of the stiff neck of the child with poliomyelitis. In some of the cases of acute general myofibrositis a lymphocytosis had been found in the cerebro-spinal fluid, and in some of these cases a diagnosis

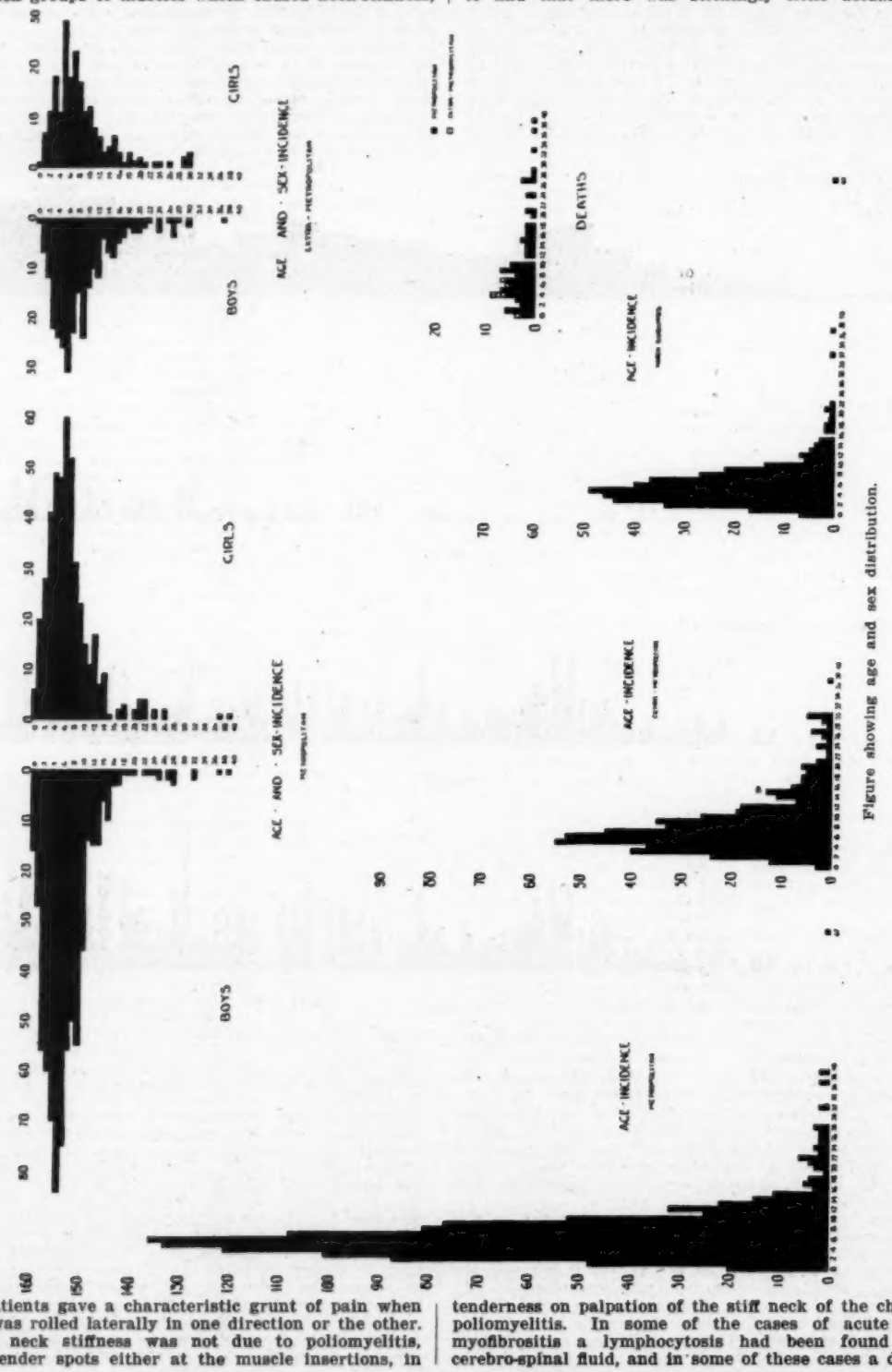

Figure showing age and sex distribution.

of atypical poliomyelitis had been made, but Dr. Powell had formed the opinion that though they might be of virus origin the virus was certainly not that of poliomyelitis.

Mental clouding and convulsions were the next subjects discussed in this connexion by Dr. Powell. Two patients only in the series had been so stuporose as to be unable to answer questions. The stuporose condition, which was very rare, did not last long; by the next morning the patients were as a rule rational again. Mental clouding was much more commonly seen in meningitis. Dr. Powell contrasted the unseeing gaze of the child with acute meningitis and the obviously intelligent eyes of a sick baby with poliomyelitis with a very stiff neck and back. Convulsions had not occurred in the series, but it was well known that they were extremely rare in poliomyelitis.

Dr. Powell stated that a rigor had not occurred in any of the cases and must be distinctly unusual and that, apart from coincident impetigo or scabies, a rash of any description was usually indicative of a condition other than poliomyelitis.

Dr. Powell then referred to the consistent absence of splenic enlargement of appreciable extent in poliomyelitis. On the other hand, the spleen was palpable very commonly in many of the other infections in childhood. The acute throat and respiratory infections, bronchopneumonia, the bowel toxæmias, catarrhal jaundice, tuberculous abdominal conditions, tuberculous meningitis, and even severe impetigo were all frequently accompanied by sufficient enlargement of the spleen to render it palpable. He had seen only three definite cases of poliomyelitis associated with this degree of splenic enlargement; in two instances this feature was explained by the recent occurrence of severe impetigo, and the third patient had had a severe local infection in the leg with enlargement of the lymphatic glands. The presence of a readily palpable spleen was pronounced by Dr. Powell to be distinctly against the diagnosis of poliomyelitis.

At this stage of his address Dr. Powell reverted from the negative to the positive consideration of the signs and symptoms of poliomyelitis. In his search for symptoms which could be classified as truly diagnostic of the disease he had failed, but there was one symptom which was highly suggestive if volunteered by the parent or could definitely be established to have been present. The symptom to which he referred was a coarse intention tremor which was usually referred to by the parents as a tremble in the hands or the cause of shakiness which had resulted in the dropping of cups or other articles. The fine tremor of other acute infections was not infrequently noted by parents, but the more coarse intention tremor of poliomyelitis was commented on by them probably because it was more noticeable as it was seen at its maximum on voluntary movement. Dr. Powell stated that because he considered they were of very great importance he always asked for three symptoms, diarrhœa, cough and tremor, if information about them was not volunteered during the taking of the history.

With reference to lumbar puncture, he said that he had been very surprised at the limitations of its use as a diagnostic procedure, and he summarized the position under five headings: (*a*) At least 90% of the cases were diagnosable without lumbar puncture and in these cases the findings on examination of the cerebro-spinal fluid were merely confirmatory. (*b*) In a very early case of six to eighteen hours' duration the cerebro-spinal fluid findings were very often normal and the old idea that a preceding meningitis occurred could not be upheld. (*c*) The absence of cells in the cerebro-spinal fluid could not be regarded as indicative of the absence of poliomyelitis, and conversely an increase of lymphocytes and globulin did not necessarily prove the presence of poliomyelitis, for exactly similar findings might be made in the early tuberculous meningitis or when a cerebral abscess was present. (*d*) It was conceivable that a lymphocytosis occurred in the cerebro-spinal fluid in many common conditions in which it was not usual to perform lumbar puncture. Dr. D. O. Brown had mentioned an instance to Dr. Powell in which that fact had been established in association with osteomyelitis of the ilium. (*e*) Very occasionally, however, suspicious findings in the cerebro-spinal fluid had been the first evidence of poliomyelitis in certain cases in which that diagnosis was thought unlikely, but was subsequently confirmed to be correct. Dr. Collins, Medical Superintendent of the Children's Hospital, had had a recent example of the truth of this point.

Dr. Powell considered next the cases that had been most difficult to assess. He thought that the screaming, uncooperative, nervous child or baby in the early stages of the disease could be difficult to assess to the point of distraction. These children were all stiff in the neck and back. They would not or could not kiss their knees, but at times it was very difficult to decide the extent to which failure could be ascribed to bad temper or to poliomyelitis. It was in those cases that the "navel sign" had perhaps the greatest value of all the signs. The light from an electric torch or some attractive toy was placed in the region of the umbilicus and the child was encouraged to look at it. It was not necessary for the doctor to be present near the child; the doctor from a distance could observe the attempt of the child to look at the object. The degree to which the child would get the chin onto the chest in looking at the object supplied a very accurate measure of the amount of neck-stiffness. The sign was important because it was strictly objective and the child was not conscious that a test was being applied. Another difficult type of case mentioned by Dr. Powell was the one in which the differential diagnosis between generalized myofibrositis of infective origin and poliomyelitis had to be made. The case in which facial weakness was present was also difficult. The condition might be a Bell's palsy or poliomyelitis. Study of the background usually allowed the differentiation to be made with certainty. A patient with Bell's palsy would walk up to the doctor declaring he felt perfectly well and almost joking about the twisted face; there would be no headache, vomiting or drowsiness, though there might be some pain and tenderness over the stylo-mastoid foramen on the same side as the palsy. The unilateral pain and tenderness Dr. Powell had previously indicated as a point against the diagnosis of poliomyelitis. Apart from the palsy, the patient would be afebrile and would look the picture of health. On the other hand a seventh nerve palsy in which the weakness could be as complete as that of Bell's palsy, occurring with the other features of virus infection in a patient who was obviously ill with poliomyelitis, did not present any difficulty. It was in some of the mild cases that trouble in diagnosis was experienced. There was, however, almost always a background of headache, some drowsiness, lassitude and possibly fever which had been going on for several days; the patient might show some slight neck stiffness or difficulty in kissing the knees, or by careful examination some muscle weakness elsewhere might be found. Dr. Powell summed up by stating that with Bell's palsy there was usually a history of abrupt onset of the palsy in a patient otherwise well and that the facial weakness of the patient with mild poliomyelitis was of insidious onset and that the patient at the time was slightly but definitely ill. He also stated that there was no need to consider other causes of seventh nerve weakness as the two mentioned were by far the commonest in the present connexion.

Dr. Powell had illustrated the points in his remarks with lantern slides, and he also showed a cinematograph film which depicted him examining patients during the present epidemic. The film could be followed quite clearly, and it served admirably to point out the exact meaning of the methods he had described for establishing the diagnosis.

The Work and Problems at the Infectious Diseases Hospital.

DR. F. V. G. SCHOLES, Medical Superintendent of the Infectious Diseases Hospital at Fairfield, after expressing his appreciation of the informative and remarkable addresses of Dr. Dale and Dr. Powell, stated that he proposed merely to speak of the work done and the experiences at Fairfield in the present epidemic of poliomyelitis. He summarized the position by announcing that up to that day 1,009 patients had been admitted to the poliomyelitis wards since the epidemic began in June, 1937. In 891 of the 1,009 cases the diagnosis of poliomyelitis had been confirmed. Fifty-six of the patients had died and 637 had been transferred for after-treatment or dis-

charged to their homes. In 118 cases the diagnosis had not been confirmed as poliomyelitis.

Dr. Scholes proceeded to the description of the accommodation and management of approximately 900 patients and the problems associated with the work. The hospital at Fairfield was just over the autumnal wave of diphtheria when the epidemic of poliomyelitis commenced. Scarlet fever and pertussis were quiet and measles was absent. Dr. Scholes had been able to turn over two ward blocks for poliomyelitis right away and gradually nine ward blocks had been taken over for the purpose. The first difficulty was that patients with other infectious diseases still had to be admitted to hospital. He had taken the administrative step of restricting the numbers of these patients admitted, though it had turned out to be unnecessary, as the incidence of diphtheria and scarlet fever had dropped to a remarkable degree. No difficulty had been encountered in accommodating the patients with poliomyelitis; they had been spaced well and the wards had been staffed with the normal nursing staff of the hospital. Some thought was needed in the selection of the nursing personnel. He considered that the nurses who went into the poliomyelitis ward did a plucky and devoted thing; they did not know that they were not running a risk and the death rate for adults was at that time very high, yet they did it without a murmur or a whimper. He and the matron had decided that the nurses who went into the ward for acute cases should be those who had had five years' experience of the nursing of children and preferably that this nursing should have been done in Melbourne. Fortunately there was a large number of nurses at the hospital who had been there for many years, and at the present time over ninety nurses were employed in the care of the patients of the epidemic. The ward blocks had been opened one by one. Half of a fifty-bed subdivisible block had been reserved as an observation division for the patients suspected of poliomyelitis who had turned out to be suffering from all manner of conditions, including rheumatism, broken neck and hysteria. The nurses were informed that they could wear masks or not as they pleased in the poliomyelitis wards, and very few of them had chosen to wear masks. On the other hand the nurses in the other wards who were looking after patients with diphtheria, scarlet fever or other infectious diseases were held to be in possible danger, as some of them were younger and less experienced and might pick up the infection from unsuspected quarters; it had been made compulsory for these nurses to wear masks when attending their patients. Dr. Scholes said that with the progress of the epidemic they had gradually sorted out the ward blocks into: admitting wards of which there were two or three in which the diagnosis was established and the precise amount of damage to muscles was assessed; convalescent wards in which the splints were fitted before the patients were transferred for after-treatment; the observation wards to which he had already referred; and the respirator wards. In all the wards for acute cases none but seasoned and immune nurses had been employed, and the same applied to the observation wards. In the convalescent ward some younger nurses had been required to complete the complement. The medical staff had consisted of four resident medical officers who had been told off for the whole duties of the poliomyelitis wards, which included admission, assessment of disability, all the arrangements concerning splinting, and the selection for transfer for after-care.

Dr. Scholes referred to the special provision of respirator boxes for respiratory paralysis as a new feature in this country. Hitherto it had been necessary to sit by and watch victims die, but that was all altered. If the paralysis was only respiratory and temporary the patients could all be saved, but extension of the paralysis to pharyngeal or circulatory centres often rendered the artificial respirators insufficient to save life. It was well known that the patients were put into improvised boxes after the fashion of the Drinker respirator, but that the mechanism for keeping up the artificial respiration was not identical with the Drinker apparatus. They were greatly indebted to Professor Burstal, of the Faculty of Engineering at the University of Melbourne, for the efficient manner in which he had supervised the construction and erection of the respirators. The number in use had increased from time to time. They had commenced at one end of the ward, and up to the present twenty-three respirators had been provided and occupied the whole of the ward. Forty or fifty patients were receiving respirator treatment at the time of the meeting; some of them had to be in the boxes all the time, but others needed the treatment for only three or four hours daily to give the muscles of respiration rest and support. It was indisputable that many a life had been saved by the respirator treatment, but the problems associated with it were manifold. These problems were the subject of a paper by Dr. McLorinan and Dr. Watson published in THE MEDICAL JOURNAL OF AUSTRALIA. An ingenious gadget for the administration of oxygen had been evolved as a solution of the problem of cleaning, changing and sponging children who could not do without the respirator even momentarily. Dr. Scholes also referred to the difficulty of applying reasonable movement for the prevention of deformity of the patients in the respirators. Several of them had sustained pharyngeal paralysis and dreadful disabilities of the limbs. The masseuses had done wonderful work for them, but that problem was still unsolved. He stated that in a number of children diphtheria organisms were present on the nasal mucous membranes, and when one of these children had to go into a respirator it was very difficult to know what was the best course to adopt. Four of the patients in respirators had continued to give positive swabs. Twenty-six patients who had required respirator treatment had died, most of them from pharyngeal and respiratory paralysis; but there had been some occult causes of death. Doubtless in some cases involvement of the cardiac centres in the central nervous system was a factor, but pharyngeal paralysis was the great cause of death.

Dr. Scholes then spoke of the provision of splints and the attendance and work of the masseuses. He said that the patient's temperature came down in a few days as a rule; the disability could then be assessed and the degree of absence of paralysis established. No further extensive paralysis was likely to occur, and at that stage it could be decided what splinting was necessary. Right from the beginning there had been close cooperation between the hospital at Fairfield and the Children's Hospital and it had been a big job; over 600 splints had been made by the workers in the splint-making shop at the Children's Hospital for patients in the hospital at Fairfield. This had necessitated daily visits by the splint makers and collaboration with the masseuses in measuring and fitting and attending to the splints. By the time this work was completed the patient would be ready for transfer for after-care. An assessment was made at the hospital in the third week on medical grounds to reach a decision concerning the suitability of each patient for transfer to an institution or to his home. This decision was notified to the After-Care Committee which inquired into the home conditions and circumstances. It had been found necessary when the home conditions were unsatisfactory to make provision in after-care institutions for some of the children who on medical grounds could have been managed at home. He said that the hospital at Fairfield had provided all the transport for the patients; an ambulance was sent for each patient prior to admission to hospital, and the patients were transferred on discharge from hospital to the next destination in similar fashion.

Dr. Scholes then spoke of the very varied assortment represented by the 120 cases in which the diagnosis of poliomyelitis had been unconfirmed. They had included abdominal conditions of which the commoner were appendicitis and pyelitis; acute pneumonia; tonsillitis, with involvement of the cervical glands; rubella, with enlargement of the occipital glands; rheumatic conditions, both fibrositic and ordinary rheumatism of the juvenile type; osteomyelitis; various forms of injury; a meningeal group, including three verified examples of tuberculous meningitis; one or two patients with encephalitis; and others with vague illnesses. The borderline cases had caused a great deal of concern; patients with fever, vomiting and headache associated with stiffness and pain in the

back of the neck, in the cases of which no definite diagnosis could be made, had been admitted at times to the poliomyelitis ward for observation. Some of them doubtless had poliomyelitis, but were not reported as such and were not paralysed. Lumbar puncture had decided the point in a few of these borderline cases which had ultimately been diagnosed as poliomyelitis and reported. They had recognized abortive poliomyelitis in some 50 of the 891 reported cases of poliomyelitis; this proportion was less than was usual in epidemics and the authorities could not be accused of over-estimating in that fashion the numbers of sufferers in the epidemic. The abortive disease had been diagnosed on clinical grounds supported by the results of lumbar puncture or alternatively by the signs and symptoms combined with the history of contact in the house or family between ten and twelve days prior to the onset. It had been decided in those abortive cases to watch for the late development of paralysis, but the latest development had occurred about twelve days from the beginning of the symptoms.

The paralysis had occurred more commonly on the fourth, fifth or sixth day after subsidence of the fever, and in some cases was moderately severe, but the later paralyses had invariably been mild.

Information likely to be of interest to hospital administrators was given by Dr. Scholes. He said that in any hospital with a large turnover it was inevitable during an epidemic that some patients incubating the disease would be admitted to hospital with some other condition. They had experience of five such primary cases, two patients with scarlet fever, two with diphtheria, and one who had been admitted as suffering from laryngeal diphtheria. The last-mentioned patient was quickly recognized to have had laryngeal paralysis and was transferred to the poliomyelitis wards before the subsequent development of paralysis of some of the trunk muscles. Only two secondary cases had resulted. A patient with occult scarlet fever continued to have a raised temperature for a little longer than usual and had had vomiting and tremor; he turned out to be suffering from poliomyelitis as well as scarlet fever. A patient in the next bed was infected by him, and it was then found that the infector came from an area affected by poliomyelitis, and four weeks later a playmate of that patient was admitted to the hospital with poliomyelitis. The patient with the secondary infection was transferred to isolation long before the paralysis developed. A nurse who had attended him had a vague illness and went off duty; fourteen days later a child with pertussis who had been attended by the nurse developed paralysis. Thus within a period of approximately fifty-six days the accidental admission to hospital of one patient with abortive poliomyelitis had caused two patients to be paralysed and one other person to have the disease in the abortive form. Those were all the cases they had had at the hospital in Fairfield.

Dr. Scholes then referred to two future problems: that of the patients requiring continued respirator treatment and that of the very severely paralysed patients. He said that 673 of the patients had been discharged from the hospital and that approximately 200 remained. Thirty or forty of those who remained could be termed the dregs of the epidemic; they were the most severely affected. Some of them were children who had been in respirators for three, four or five months and might never come out of them alive. Others who had required respirator treatment for two or three months could remain out for only two or three hours at a time. Some were shockingly paralysed, and he could contemplate the future for those children with nothing but dismay. After all, they represented only a small proportion of the patients affected, and recovery had been more rapid than in previous epidemics. They would like to feel that the work done for the patients at the Infectious Diseases Hospital in the initial three weeks could be related to the gratifying speed of recovery and the low death rate. He had been touched by the kindly remarks along these lines that had been made to him. He was very proud of the hospital at Fairfield and was gratified by praise given to it by previous speakers.

Dr. Scholes added later that pharyngeal paralysis had been the great cause of death, with or without respiratory paralysis. As in post-diphtheritic paralysis, death occurred from bronchopneumonia or dehydration with rapid emaciation and collapse, but in poliomyelitis there was a possible additional factor, associated cardiac paralysis. Complete pharyngeal paralysis was almost always, possibly always, fatal, but there were severe cases in which life could be saved, and many more mild and partial cases in which the outlook was good, with efficient hospital treatment. In these last, as with similar forms of post-diphtheritic paralysis, recovery was usually rapid. Long experience by the nurses in this form of paralysis had made possible a quite notable saving of life in the present epidemic.

While no doubt much good had been done by the early admission of patients with ordinary forms of poliomyelitis to hospital, Dr. Scholes thought that it was in the pharyngeal cases above all that early treatment in hospital was imperative. Life could be saved in moderately severe and complications minimized in the milder cases.

Problems of After-Care.

DR. JEAN MACNAMARA, in speaking of some of the problems of the after-care of poliomyelitis patients, said that Dr. Scholes had already outlined the part played by the Fairfield Hospital in the first stages of after-care of paralysed muscles when he had referred to the assessment of the degree and extent of paralysis and the measurement and fitting of suitable splints. She said that she would like to add her personal tribute to the wonderful work which had been done at Fairfield and stressed the quiet efficiency with which the details had been carried out. After the discharge of a patient from the hospital at Fairfield, either to his home or to an after-care institution, his care rested with the After-Care Committee and its staff. The provision of efficient care to 100 paralysed patients was relatively simple; to 1,000 children much more difficult; but the difficulties were those of administration and organization rather than of knowledge or policy. She thought that it was a pity that the accumulated experience and wisdom of men who had spent their lives studying a problem were often overlooked, and she regretted the tendency of many to test some bright ideas of their own and not to learn from the experience of others. In the study of the recovery and care of the partially paralysed muscles it was profitable to search for the conclusions of men of long experience, because observation of individual cases over years was necessary before one was justified in forming opinions as to the value of any method of treatment. Intelligence, high degrees or hours of thought did not equip one as did years of careful watching, letting the muscles themselves teach what they liked and what they did not like.

Dr. Macnamara said that of the pioneers of poliomyelitis she owed most to Charles Fayette Taylor, who had published a small book on infantile paralysis in 1867; his principles were as true today as they had been seventy years earlier. He appreciated fully the natural tendency towards recovery of function, a fact which many of the public were only just commencing to appreciate. He had stated that all cases tended towards recovery and that very few cases were to be met with in which some portions of the originally paralysed muscles had not entirely recovered. He had pointed out that there were cases so bad that the sufferer must always remain without hope of essential improvement, but that the large majority of all the patients whom he had seen had had more than twice the paralysis when it occurred or when it was first discovered than they had at the end of the first year. Charles Fayette Taylor had stated that he thought Nature did with comparative rapidity what she could, and would often do much more if she was permitted. He had thought that many a theory had been triumphantly established by chance trial at a time when Nature was sure to be using her reserve forces to repair damage, and always with more or less success. He had held that if the apostles of electricity, strychnia and the ice bag had no better data than partial recovery in a few cases within the period of the first recuperative efforts, their claims rested on a very unsatisfactory basis. Dr. Macnamara said that

Taylor had also appreciated the factors which led to deformity. He had stated that the shortening of certain muscles was not a necessary consequence of infantile paralysis, and when it occurred was simply the adaptation of their length to the position they happened to be in. He had thought that it was entirely accidental which muscles became shortened, whether flexors or extensors; therefore deformities were not a necessary consequence of infantile paralysis, and when they were allowed to occur the process of recovery was arrested. Dr. Macnamara thought that both Taylor and Lovett had answers to a question which was a live one today. She had heard it said repeatedly during the prevailing epidemic that if physical therapy and muscle reeducation were good, it must follow that more and more muscle reeducation would be still better. It had also been stated that if one granted that a weakened muscle needed exercise, it must be exercised every two hours or several times a day. She considered that the question deserved both consideration and observation. Any gardener knew that a small quantity of ammonium sulphate was good for his lemon tree; but it did not follow that still more ammonium sulphate was any better for it. He also knew that orchids required a higher temperature than other flowers, but if the temperature were trebled it would not necessarily result in larger orchids. Similarly in medicine there were drugs, such as insulin, regarded as useful in certain doses, but the administration of eight times the dose was not followed by a corresponding increase in benefit, but often by actual harm.

Dr. Macnamara pointed out that with living cells, whether human, animal or plant, there were certain optimum conditions for growth. With weakened muscles it was the task of the doctors to study what were the optimum conditions for encouraging recovery of power. They knew that maintenance of even temperature helped recovery and that stretching hindered it; the amount of exercise optimal for improvement required careful assessment for individual muscles at different stages of their recovery, but exercise could very easily be overdone. Dr. Macnamara added that Taylor had also given considerable help with the recuperative period which was a very critical one. In this phase of the disease a certain amount of recovery had taken place with the corresponding amount of improvement in some at least of the muscles. She wondered how that recuperative process could be kept up. Every organ was strengthened by the proper use of its function; whatever power there might be in a muscle, after a certain time needed to be used to develop more power, but there might not be enough for the purposes of locomotion of actual use. It was evident then that one must continue to afford the muscles an opportunity to act within their capacity, alike avoiding inactivity or over-activity.

Dr. Macnamara said that Lovett had recorded towards the end of his life how often in the past he had exceeded the "fatigue point" by too frequent muscle reeducation. He had said that it had been repeatedly observed in his private practice that power might begin to return to a very faint degree while the patient was under muscle training, and that with care that power would steadily increase; but if that muscle were exercised very gently every day the power would diminish or disappear; such muscles were exercised only once in three days, the work being very carefully increased. He had said that illustrative cases, tested with the spring balance, seemed to show that much smaller degrees of over-use might be more deleterious than were generally supposed. He had considered that it was necessary to know the dose of the remedy that was being used. The muscle test had shown the surprisingly small amount of exercise which was detrimental to recovering muscles; in some muscles returning power had been wholly abolished by over-use. Taylor had said that the advice he had often been given, to use affected limbs as much as possible, was in his opinion the worst possible advice. Dr. Macnamara said that in 1925 and 1928 tests had been carried out with the spring balance in Melbourne and they had impressed upon her how easily the fatigue point could be exceeded and how carefully the amount of exercise needed for a certain muscle at any particular time should be assessed.

Dr. Macnamara, in referring to splinting, said that the question as to whether patients with mild infections were being splinted unnecessarily had been raised. It was not easy to decide whether a child examined within two or three weeks of the onset of illness was a normal child, because at that stage the test of exercise beyond the fatigue point could not be made. The child was asked to walk, to run, to hop on each leg, to walk on tip-toe, to walk on the heels, to walk like a sleuth, very slowly on tip-toe, with bent knees and hips, to rise from the sitting position on both legs and then on one at a time. If in any of these tests a deformed position was assumed, it was evidence of weakness and it was safer to use splints. For example, a knee might hyperextend, a foot evert or invert, the pelvis might dip, or it might not be possible to make the tests, or they might not be performed as well on one leg as on the other.

If any weakness was detected in the trunk or lower limbs, even if the weakness was slight, it was safer to apply a splint to a little active child, using the splint as a means of regulating the return to activity and of ensuring good posture in the intervals between activity. Dr. Macnamara said that at times during the year they might occasionally have played too safely, but, on the other hand, there had been controls: children had been kept at home; children had been regarded as suffering from too mild or too indefinite an infection for them to be sent to hospital; children had been allowed to resume full activity as soon as they felt ready for it. A number of those children had been brought up to the hospital later because of limping or scoliosis. A mother had sometimes stated that her child walked perfectly well when he got up in the morning and commenced to limp only after he had been walking some time. Surely that statement was a clue as to what those muscles needed—exercise up to but not exceeding the fatigue point; and its determination could not be left with safety to the inclination of a child four years of age. A splint such as the modified double Thomas splint which was being used, enabled activity to be graded to the amount of exercise needed.

Dr. Macnamara also said that some doctors might wonder why a child with involvement of the muscles of only one shoulder should leave hospital in a complete splint with arm-piece attached. She pointed out that at the time of the examination to assess muscle weakness, two or three weeks from the onset, it was impossible to say with certainty that the trunk muscles were perfect. Moreover, even if they were, the asymmetrical load of a deltoid splint tended to induce a scoliosis. The scoliosis that had been seen in the past, which had developed following the early application of the deltoid splint, described first by McKenzie, had led them to postpone the application of the ambulatory deltoid splint for some weeks, and then to allow the child wearing it to stand and sit, and a little later to resume walking, teaching him at first in front of a mirror what was the straight position, watching for any tendency of the back to curve, and using the big modified Thomas splint with arm-piece to hold his trunk straight and the shoulder abducted all night and when he was resting during the day.

In conclusion, Dr. Macnamara referred to the training of the staff of physio-therapeutists. She said that they had had in Victoria a series of small rehearsals for the present epidemic. The outbreaks since 1925 had attracted into the massage or physiotherapy profession a fine type of girl. Each epidemic had added to the knowledge and experience of the physio-therapeutists who had devoted their whole time to the care of paralysed muscles. In the last analysis the results of the present epidemic would depend on the work carried out by those girls. She asked any practitioners present at the meeting to help them and to appreciate their knowledge, to collaborate with them and to give them a chance to show what results they could achieve. She asked them to do that before testing any new ideas, such as the hourly treatments, application of complete plasters or the use of stationary bicycles. She wished the practitioners to

realize that those experienced girls had spent every day for eight or twelve years caring for muscles as a good gardener cared for delicate plants. She did not think that any doctor could feel humiliated by admitting that he could learn from specialists in that province.

Dr. Macnamara added that they were aiming to send experienced people to the country centres. It was essential to have girls of wide experience on the itinerant work, caring for children in the homes. To do this it might be necessary to limit the number of experienced girls in institutions to one in charge, teaching interstate physiotherapeutists who had come over to help in the epidemic. She concluded by saying that she hoped that the practitioners at the meeting that night would do their best to help those girls; their work was very hard, their lives were rather lonely and their enthusiasm was great.

DR. J. B. COLQUHOUN spoke concerning some of the problems of after-care which had arisen in his experience of the work as one of the after-care medical officers. Though he said that one of the problems was provision of an adequate staff of physio-therapeutists for muscle reeducation, he paid a tribute to the way in which Dr. Jean Macnamara had trained women for this work during the past ten years and said that the work would have been beyond him had these women not been available. He spoke also of their efficiency, and said that he had learned a great deal by discussing the clinical features of the individual patients with them. If a home had a garden or a veranda most of the patients could be treated there; physio-therapeutists went into the homes and the service was greatly appreciated by the parents. If any were not satisfied he had not heard of it; he had yet to come across one who would say so to his face. The children were being looked after very well and were as happy and as well treated as they could be under any other system. One of the difficulties, of course, was that the best physiotherapeutists were being sent off to the country and to the itinerant jobs, but Dr. Jean Macnamara had seen to it that at least one good one was left behind in each of the after-care institutions to train the less experienced physiotherapeutists. These locally trained women compared more than favourably with members of the massage staff who had come from other States. Although otherwise highly qualified, hardly one in five of those visitors was capable of making a muscle examination, and at times the medical officers had had to commence by teaching them the anatomy of muscles. He thought it was improbable that trained nurses could be rendered efficient at that class of work after so short a period of training as six weeks.

Another problem was that of the supervision of the attitude of a patient at rest in a splint. Dr. Colquhoun had worked in Lovett's clinic and could say without fear of effective contradiction that the physio-therapeutists trained in Melbourne compared well with the very best in the Boston clinic. He expressed the conviction that splinting was absolutely necessary to keep paralysed muscles at rest, and that in the early stage after the onset of the paralysis general splints should be applied. The community owed a debt of gratitude to the Children's Hospital for the splint department in addition to the physio-therapy department. Two years earlier four people had been employed in the splint shop, but now there were fourteen or fifteen and only two of them were not cripples. The shop was a hive of industry, and it was gratifying to see a workman making an honest living at a skilled trade instead of receiving a pension and leading a life of enforced idleness.

The general education of the patients through the years of treatment that would be necessary for those more severely affected was a problem that had yet to be tackled adequately. Many kindergarten helpers visited the children and assisted in the work to save the time of the skilled personnel when possible. They also helped to keep the children happy and performed little services of a kindly nature such as inquiring after the progress of some of the mates of the children they attended. Dr. Colquhoun felt that the important part of his own work was to back up the physio-therapeutists so that the system got a fair chance. It was helpful to encourage the parents to have confidence so that all working together could carry out the principles for which they stood. He asked that if outside practitioners were consulted about any of the patients, they should be guarded in the way they expressed the prognosis as to the duration of the paralysis or the progress that should be made by the patient. It was true that half the patients would make rapid progress and would soon be able to be relieved of the splints, but when key muscles were affected the duration of splinting and of treatment would be greatly prolonged. Differing opinions confused the parents and discouraged the patients and might lead to unsatisfactory cooperation in after-treatment.

With reference to correction of deformity, Dr. Colquhoun considered that the requirement would be small in those patients who were followed up closely by the after-care officers; but he thought that certain so-called abortive cases in which patients failed to cooperate and the cases of certain patients perhaps from remote parts of the country who failed to receive the benefit available to those in larger centres, would swell the ranks of those who in later years would require corrective treatment. Dr. Colquhoun referred to the skilled judgement which was required to decide the appropriate time and the appropriate type of corrective treatment, and took the opportunity to say that people with adequate training were available to deal with this class of work and that it was the type of work which should be referred for the purpose. By splinting and by surgery even the severely damaged victims of the epidemic would ultimately be capable of rehabilitation. Some children at the Frankston Orthopædic Branch of the Children's Hospital did very good handwork. It would be necessary to set up an after-care scheme to review the badly paralysed patients from time to time. Medical practitioners were requested not to say that nothing could be done for those patients without consulting the Society for Crippled Children, who would make an earnest attempt to overcome all the difficulties. He considered that it was of paramount importance between epidemics to maintain a staff of highly skilled physio-therapeutists and a skeleton organization for the manufacture of splints so that the lamps could be kept trimmed at all times in case a serious epidemic developed.

Dr. Colquhoun spoke of the problem presented by the length of time spent in visits to the patients in private homes. He found he could attend to only four or five patients in an afternoon if he visited them at home. He had been forced to start clinics at Elsternwick, at South Melbourne and at Ormond. At Elsternwick and at Ormond the clinics were held once monthly and at South Melbourne twice a month. All-day clinics were urgently needed at South Melbourne and at Port Melbourne. He asked medical practitioners to use their influence to see that those suburban clinics were continued. A visit to the clinic was an outing for the patient, and it was good for the children to see new surroundings. The mothers had a difficult job, and it was a welcome break to them to converse with each other about the patients. He found that the mothers were helpful to each other in commenting kindly on the progress made even though it seemed small. Encouragement of the parents was very important, as it meant at times that they renewed their efforts when otherwise they might lose heart.

In conclusion, Dr. Colquhoun spoke of a girl who had come under notice because she was unable to do her work at a factory satisfactorily. Examination showed that one deltoid muscle and the opposite *opponens pollicis* were the only muscles paralysed. With rest the deltoid muscle had recovered from the degree of weakness recorded as 3 to the degree recorded as 1, and with adequate care she would not lose her job and would be able to resume her work as a packer. Dr. Colquhoun stressed the fact that inaccuracy in diagnosis of the minor degree of paralysis and improper treatment would have harmfully affected the whole of that patient's future life.

A MEETING of the New South Wales Branch of the British Medical Association was held at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, on November 25, 1937, DR. L. A. DEY, the President, in the chair.

Splenectomy.

DR. LORIMER DODS read a paper entitled "Indications for Splenectomy in Pædiatric Practice" (see page 377).

DR. A. T. NISBET first discussed Banti's disease. He said that of all the ailments attributable to the spleen, Banti's disease was the one which at the moment appeared most interesting. During the last few weeks he had combed the literature for some admission as to when and where someone had been bold enough to apply intensive X ray therapy in this disease. Nowhere could he find that anyone had committed this apparently well-justified experiment. Dr. Nisbet went on to say that two patients with this condition had been treated in Sydney and that they had both done very well. One was a man, aged eighty years, who was seen a few days before the meeting. He appeared in excellent health, his digestion was good, he could walk as far as he liked, he slept well; in fact the only thing that upset him at all was some slight trouble with his prostate gland. When he was examined, a large, hard spleen was found, extending almost to the umbilicus. It was not painful on palpation and it was considerably smaller than when he had first been given X ray therapy in January, 1935. Notes made at the time described the spleen as filling almost the whole abdomen, the notch being palpable below McBurney's point.

The blood count in January, 1935, revealed that the red cells numbered 3,170,000, and the leucocytes 6,360 per cubic millimetre. The hæmoglobin value was 49%. Of the leucocytes, 16% were neutrophile cells and 80% were lymphocytes. During the week in which the meeting was held a blood count revealed that the red blood cells numbered 4,200,000, and the leucocytes numbered 3,500 per cubic millimetre. The hæmoglobin value was 55%. The percentage of neutrophile cells had increased to 68% and that of lymphocytes had dropped to 32%.

Dr. Nisbet pointed out that this patient's illness had been more than held in check for almost three years, and that he had had the minimum amount of irradiation. In addition, no operative risk or the discomfort attaching thereto was experienced. The diagnosis of Banti's disease could naturally be queried on the high percentage of lymphocytes; on the other hand, the lymphocytes disappeared after the first blood count and the first X ray treatment, and at no time since had they been in such evidence. Also lymphocythæmia did not act like this or remain so completely quiescent over this number of years.

Discussing Gaucher's disease, Dr. Nisbet said that it was discovered in the eighties, and in spite of its rarity two patients who had been classified by senior physicians of Sydney as suffering from this condition had been treated. The one that responded the more satisfactorily lived almost two years. One other patient under treatment at the time of the meeting appeared to fall into this category, with large spleen glands, a mild leucopenia *et cetera*. In this case the clinical history showed that the spleen was the first gland to show signs of enlargement. This was preceded by a month's pain and discomfort under the left costal margin. The patient was regularly and carefully examined by her local practitioner for a month before the spleen was noticed to be becoming palpable below the costal margin. Cases of Gaucher's disease had been reported in which irradiation had been given and the patient had improved so much that splenectomy had then been performed. Dr. Nisbet said that if a newgrowth was considered inoperable at any one stage, then he considered that it was always inoperable.

Hæmorrhagica purpura seemed always much more a symptom than a disease, Dr. Nisbet said, but according to the hæmatologists it was a very definite entity. Five patients had been treated with varying results. In one the purpura cleared up in a few days and remained clear; in another the patches disappeared, but returned some weeks later; one patient did not respond to treatment and the other was lost sight of. In none of these cases, however, was it absolutely certain that the purpura was not secondary to some other lesion.

Primary newgrowths of the spleen, although classified as small round cell or large round cell endothelial sarcoma, were so extremely rare that there was no need to discuss them at the meeting.

Dr. Nisbet went on to say that the large spleen in menorrhagia was interesting from the radiotherapeutic standpoint. Women in the twenties and thirties, with profuse and continued uterine hæmorrhage, were a problem to all medical practitioners; and neither hysterectomy nor intensive irradiation was conducive to future childbearing.

About four years earlier workers began to experiment seriously with X ray therapy to the spleen, and during that period this form of treatment had been given in several of these cases. The results were extremely interesting, in that with very small doses of irradiation applied to the spleen it did appear possible to control to a very great extent this disordered menstrual flow without causing amenorrhœa and consequently sterility. Treatment had to be given very carefully and only very occasionally, and the patient had to be continually watched and possibly treated over a number of months, in order that the menstrual flow could be closely regulated and brought back to normal without giving that "little bit too much" which would cause an artificial menopause.

In leuchæmic patients with an enormous spleen Dr. Nisbet said that he thought the less irradiation the spleen received during a course of X ray therapy, the longer the patient would live. In this disease, if the thorax or the spine was given moderately small doses of irradiation, it was found that within a few days the white cells had decreased in number whilst the red cells had increased; and, more extraordinary still, the spleen had diminished in proportion to the amount of irradiation applied. The advantage of this form of treatment was that even in these days, despite advanced and modern technique, if X rays were applied directly to the spleen, they tended to make the organ very hard and fibrous, until the day was reached when so little sensitive tissue was left in the spleen that the response to irradiation became infinitesimal and of little therapeutic value.

In polycythæmia enlargement of the spleen was found, but red blood counts of 8,000,000 and over could be brought down by irradiation, not of the splenic region, but of the long bones. Two patients reported in 1934 were alive and well at the time of the meeting, and required radiation therapy only about once every twelve months. It was interesting to note that in this disease the skin was extremely sensitive to any form of X or radium radiation, and a dose of one half that usually given to people with a lower red cell count would cause an intensive erythema with moist desquamation.

Metastatic involvement from newgrowths in other organs was not so uncommon, the spleen being involved in cases of lymphadenoma, lymphosarcoma and carcinoma of other organs, such as the breast. In conclusion, Dr. Nisbet said that the question of the advisability of giving any irradiation to a spleen with secondary involvement had to be considered in each individual case.

DR. P. L. HIPSLEY said that he thought that the discussion should have been opened by a physician, because physicians probably had better knowledge of the conditions under discussion. He referred to the views of Bartlett, who gave certain criteria for splenectomy. These were splenomegaly, secondary anæmia and the failure of the patient to manifest improvement after repeated blood transfusion. Dr. Hipsley thought that if these criteria were followed clinicians would not go very far wrong. He had had several cases referred to him by physicians, skilful men, who were unable to give the disease a name. Dr. Hipsley thought that if a child had a large spleen and anæmia, and if it did not respond to repeated blood transfusions, splenectomy should be considered, whether it was possible to give the disease a name or not.

There were certain diseases in which splenectomy was indicated. In acholuric jaundice splenectomy would relieve the anæmia. It was necessary, however, to perform the operation at an early stage. If operation was

postponed for some years the gall-bladder became infected and other complications occurred. Splenectomy was not a new operation in this disease. Spencer Wells had performed splenectomy on a child with acholuric jaundice in 1887. Dawson had reported that this patient was alive a few years ago. Dawson stated that operation should be undertaken early and that it was an advantage if blood transfusion was given. The transfusion should be given after and not before operation. Another disease in which splenectomy might be useful was thrombocytopenic purpura. He had seen one or two cases of the chronic variety in which splenectomy had been successful. Splenectomy would not, however, invariably bring about a cure, and, moreover, the child might get well without an operation. Dr. Hipsley referred to a child of six who had suffered from hæmatemesis and a large spleen. Splenectomy had been advised and refused, and the child was now normal. Another condition for which splenectomy was advised was Banti's syndrome. Splenectomy did not always effect a cure, but would generally bring about improvement. It was necessary to perform the operation before the occurrence of cirrhosis and ascites; it should not be performed when the platelet count was high or when thrombosis was present.

If performing the operation on children, surgeons would find that adhesions were not numerous. An incision was made parallel to the ribs, the pedicle of the spleen was clamped with stomach clamps, and interlocking sutures were used.

DR. E. H. M. STEPHEN said that he considered Dr. Dods's excellent address left nothing he could add except a few remarks as to the frequency of some of the conditions he had mentioned. Simple purpura was not uncommon and responded readily to rest and tonics. On the other hand, of Werlhof's essential thrombocytopenic purpura he could recall three cases in his beds in the last six years. In the first case gangrene of the foot necessitating amputation had occurred; the boy recovered. The second case was similar to the first and had terminated fatally. In the third case the patient was a girl of three years with bleeding from the vagina and from the ear, and with hæmaturia. This child recovered after blood transfusion. In the same period he had seen three cases of von Lederer's anæmia. Acholuric jaundice, in his experience, was somewhat more common in hospital practice than either of the other conditions which he had mentioned.

DR. A. S. WALKER remarked that the conditions were interesting because so little was known of them. What they wanted to know was why the spleen was affected, though not unnaturally they were more concerned with what they should do when they were confronted with a case suitable for splenectomy. Dr. Hipsley had said that it was difficult to name the disease; Dr. Walker thought that the difficulty lay in explaining the disease by giving it a name. He agreed that in acholuric jaundice operation should be performed early. As far as thrombocytopenic purpura was concerned, it was difficult to make up one's mind. Who could say whether a case was mild? If the condition was obviously severe, there was no doubt as to what should be done. Dr. Walker wondered whether in doubtful cases irradiation of the spleen would take the place of splenectomy. He did not think so, but would like to hear the views of the radiotherapists. He wondered whether any of those present had any experience of the liability of patients to succumb to infection after splenectomy; at the same time he thought that this was probably a minor question. In conclusion Dr. Walker referred to blood crises in acholuric jaundice. He did not know of any disease in which the patient could look more like dying and then recover without treatment.

DR. E. W. FRECKER said that he had expected to hear a discussion as to the relative merits of surgery and X rays in the treatment of splenic affections. Dr. Dods, however, had cut the ground from under the feet of the radiotherapist by choosing for particular discussion three splenic diseases in which radiotherapy was useless.

Dr. Walker had asked whether radiotherapy could take the place of splenectomy. Dr. Frecker's answer was "no", though in certain cases it might be beneficial. Radiation decreased the size of an enlarged spleen by destroying the accumulated pathological cells therein, but probably could not correct any perverted function of the organ. As radiation rapidly caused a leucopenia, care was necessary in administration, and only small doses should be given, controlled by frequent counts of the white cells. Radiotherapists should realize that their methods were only adjuvant and not independent of other medical and surgical procedures.

Dr. Frecker did not agree with Dr. Nisbet that radiation might cause fibrosis of the spleen, as the doses applied were usually only about 100 *r* per treatment and were far too small for such a radical change.

DR. C. DE MONCHAUX said that the subject was shrouded in empiricism. Splenomegaly was obviously the common denominator in the conditions under discussion. He asked why the spleen became enlarged. Was it a cause or an effect of the disease? Until they knew the real reason behind splenomegaly, its treatment could not be very rational. Irradiation of the spleen affected either the splenic tissue itself and/or the blood and its cellular contents while flowing through the organ. And irradiation in most cases reduced splenomegaly, but the rationale of this was by no means clear. Splenectomy, of course, abolished splenomegaly effectively; but it might be important to have a spleen, and unfortunately there were no spare splenic parts, and even if there were they could not readily be incorporated in the bodily machine. He did not agree that Banti's disease was an indication for radiotherapy; rationally in fact the usual syndrome in this disease, of anæmia and leucopenia, contraindicated irradiation of an organ like the spleen. But empirically the operation of splenectomy might effect some improvement in Banti's disease.

With regard to the possibility of post-radiational fibrosis of the spleen, this was not at all likely to happen if the modern technique of small doses, both individual and total, was employed. In conclusion, it was difficult, if not impossible, to dogmatize about this subject in the present state of our knowledge. Until they knew much more about the nature and function of the spleen the treatment of that group of diseases associated with splenomegaly, whether by splenectomy or irradiation, must be regarded as largely empirical.

DR. L. HUGHES discussed the onset of symptoms in thrombocytopenic purpura. He referred to a case in which a girl first noticed a few patches of purpura when she was thirteen years old, at the onset of her menstruation. Later on, she was admitted to the Royal Prince Alfred Hospital. The clinical picture was not typical of thrombocytopenic purpura. The reticulocytes had numbered 80,000 per cubic millimetre and the spleen had not been palpable. The patient had not responded to blood transfusion, but had responded to constant drip transfusion of blood. Splenectomy was performed by Dr. Poate, and so far the patient was well.

DR. NEVILLE DAVIS agreed with Dr. Stephen that the number of cases in these conditions was small. It was difficult for anyone to give a rational basis for treatment or for prognosis. At the Mayo Clinic it was held that splenectomy caused an improvement in thrombocytopenic purpura, but that the results with radiation were not satisfactory. If symptoms recurred after improvement following blood transfusion, splenectomy was undertaken. If it was undertaken at an early stage it was efficient.

Dr. Davis went on to say that he was interested to hear Dr. Nisbet discuss the effects of radiation on other parts of the body. This opened up an interesting question as to how the effect was caused: whether it was caused through the blood stream or other agents.

Dr. Nisbet replied to Dr. Walker and said that if any doubt existed splenectomy should be undertaken. He also discussed the question of resistance to infection after splenectomy. In a severe infection the general resistance of the body was lowered and he did not think that a fatal outcome could be attributed to the splenectomy.

NOTICE.

THE Medical Secretary of the New South Wales Branch of the British Medical Association has forwarded for publication the following list of books and journals which have been added to the library of the Branch.

"The Etiology and Treatment of Spasmodic Bronchial Asthma", H. Gordon Oliver (1934).
"Medical and D.P.H. Examination Papers."
"The Control of Bovine Tuberculosis in Man", Nathan Raw (1937).
"Synopsis of Obstetrics and Gynæcology" (Seventh Edition), Aleck W. Bourne (1937).
"Clinical Reviews of the Pittsburgh Diagnostic Clinic", H. M. Margolis (editor) (1937).
"Modern Discoveries in Medical Psychology", Clifford Allen (1937).
"Early Diagnosis of Malignant Disease", Geoffrey Keynes (1935).
"Clinical Miscellany of the Mary Imogene Bassett Hospital" (Volume II), Various Authors (1935).
"Essentials of Cardiography", H. B. Russell (1936).
"Favourite Prescriptions" (Fourth Edition), Espine Ward (1937).
"Clinical Contraception" (Second Edition), Gladys M. Cox (1937).
"Medical Urology", Irvin S. Koll (1937).
"Birth Control Methods", Norman Haire (1936).
"Principles and Practice of Clinical Psychiatry", Morris Braude (1937).
"Autopsy Diagnosis and Technique", Otto Saphir (1937).
"Modern Treatment and Formulary", Edward A. Mullen (1936).
"Cancer and Diet", Frederick L. Hoffman (1937).
"Social Hygiene To-Day", Henry Ernest Garle (1936).
"Latent Syphilis and the Autonomic Nervous System" (Second Edition), Griffith Evans (1937).
"Modern Psychology in Practice", W. Lindesay Neustatter (1937).
"Surgery of the Sympathetic Nervous System", George E. Gask and J. Paterson Ross (1934).
"Science and Practice of Surgery" (Volumes I and II, Sixth Edition), W. H. C. Romanis and Philip H. Mitchiner (1937).
"Operations of Surgery" (Volumes I and II, Eighth Edition), R. P. Rowlands and Philip Turner (1937).
"A Short Practice of Surgery" (Third Edition), Hamilton Bailey and R. J. McNeill Love (1936).
"Recent Advances in Anæsthesia and Analgesia", C. Langton Hewer (1932).
"Minor Maladies and their Treatment" (Seventh Edition), Leonard Williams (1937).
"The Technic of Local Anæsthesia" (Sixth Edition), Arthur E. Hertzler (1937).
"Diseases of the Nails", V. Pardo-Castello (1936).
"Diseases of the Heart" (Second Edition), Sir Thomas Lewis (1937).
"Surgical Anatomy" (Third Edition), Grant Massie (1937).
"Statistical Methods for Research Workers" (Sixth Edition), R. A. Fisher (1936).
"Treatment in Psychiatry", Oskar Diethelm (1936).
"Motives and Mechanisms of the Mind", E. Graham Howe (1931).
"Papers from the IV Medical Service of S:T Erik's Hospital, Stockholm", edited by Hilding Berglund (1937).
"The Radiology of Bones and Joints", James F. Brailsford (1935).
"Textbook of General Surgery", Warren H. Cole and Robert Elman (1936).
"Notes on Cancer for Medical Men", The Medical and Scientific Committee.
"Weight Reduction Diet and Dishes", E. E. Claxton (1937).
"A Synopsis of Hygiene" (Fifth Edition), W. W. Jameson and G. S. Parkinson (1936).
"The Treatment of Asthma", F. T. Harrington (1936).
"A Textbook of Bacteriology", Thurman B. Rice (1935).
"Diseases of Infancy and Childhood", Wilfrid Sheldon (1936).
"Kidney Pain", J. Leon Jona (1937).
"What is Wrong with British Diet?" Harry Campbell (1936).
"The Incidence of Anæsthetic Complications: Their Relation to Basal Narcosis", C. J. M. Dawkins (1936).
"Synopsis of Ano-Rectal Diseases", Louis J. Hirschman (1937).
"Textbook of Medicine" (Third Edition), edited by J. J. Conybeare (1936).
"Practitioner Handbooks: Favourite Prescriptions", Humphry D. Rolleston and A. A. Moncreiff.
"Applied Dietetics", Sanford Blum (1936).
"Malaria in Ceylon", C. L. Dunn (1936).
"The Common Cold and Influenza", J. E. R. McDonagh (1936).
"The Prevention and Treatment of Disease", William Mitchell Stevens (1936).
"The Diabetic A B C", R. D. Lawrence (1936).
"Illustrations of Regional Anatomy", Sections VI and VII, E. B. Jamieson (1936).
"A Manual of Pharmacology" (Eighth Edition), by the late W. E. Dixon, revised by W. A. M. Smart (1936).
"Exophthalmic Goitre and its Medical Treatment" (Second Edition), Israel Bram (1936).
"Diseases of Women" (Eighth Edition), H. S. Crossen and R. J. Crossen (1935).
"An Index of Treatment" (Eleventh Edition), edited by Robert Hutchison (1936).
"Diagnosis and Non-Operative Treatment of Diseases of the Colon and Rectum", G. Schwarz, J. Goldberger and C. Crocker (1937).
"Hospital Organization and Management", Malcolm T. McEachern (1935).
"Nostrums and Quackery and Pseudo-Medicine" (Volume III), Arthur J. Cramp (1936).
"The Endocrine Organs in Health and Disease", Sir Humphry D. Rolleston (1936).
"Diseases of the Nervous System" (Sixth Edition), Jelliffe and White (1935).
"Pulmonary Tuberculosis" (Volumes I and II), Maurice Fishberg (1932).
"The Australian and New Zealand Pharmaceutical Formulary" (1934).
"The American Medical Profession 1783 to 1850", H. B. Shafer (1937).
"Recent Advances in Neurology", W. Russell Brain and E. B. Strauss (1934).
"Year Book, for 1935 and 1936, of Neurology, Psychiatry and Endocrinology", Reese, Paskine and Sevringhaus (1936 and 1937).

Urologica, 1935; *Journal of Industrial Hygiene and Toxicology*, 1937; *California and Western Medicine*, 1937; *The American Journal of Surgery*, 1937; *The Journal of Physiology*; *The Journal of Pathology and Bacteriology*; *Archives of Pediatrics*; *The American Journal of Obstetrics and Gynecology*; *Journal of Endocrinology*.

NOMINATIONS AND ELECTIONS.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Hill, Bruce Goodwin, M.B., 1935 (Univ. Sydney), 517, Pacific Highway, Killara.
Meyer, Frank Graham, M.B., B.S., 1938 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Medical Societies.

PUBLIC MEDICAL OFFICERS' ASSOCIATION OF NEW SOUTH WALES.

THE twelfth annual general meeting of the Public Medical Officers' Association of New South Wales was held at the British Medical Association House, 135, Macquarie Street, Sydney, on January 24, 1938.

DR. OLIVER LATHAM occupied the chair, and seventeen members were present. Eleven forms of proxy were presented and accepted.

Annual Report.

The secretary presented the committee's report for the year 1937, which, after much discussion, was adopted. The report is as follows.

1. During the year 1937 a forward policy has been pursued, and efforts have been made to secure improved conditions in all the Government services. The immediate results have been disappointing, but much useful work has been done which should bring appreciable benefits in the near future.

2. The committee met eleven times during the year, the attendances of members being as follow: Dr. O. Latham (President), 11; Dr. W. K. Flook, 10; Dr. C. E. Percy, 9; Dr. J. Cooper Booth, 10; Dr. J. McManamey, 9; Dr. Edelsten Pope, 5; Dr. G. L. C. Saunders, 6; Dr. H. H. Nowland, 7; Dr. J. M. Rainbow, 9; and Dr. H. H. Willis (Secretary), 11. The illness of Dr. Edelsten Pope during a large part of the year is recorded with regret and sympathy.

3. There are now 103 members on the association's roll, of whom 10 are honorary, having retired from the services on account of age. There are several eligible medical officers who have not yet joined us.

4. Three circulars have been distributed to members during the year to acquaint them of the association's activities.

5. Efforts to place our retired members in suitable employment have been only partially successful. The goodwill and assistance of all our members is again requested in this difficult task.

6. No communications have passed from the Association to other service organizations during the year.

7. Our relations with the British Medical Association, never unsatisfactory, have improved markedly in recent years, and in the year just passed hearty cooperation was given us by the New South Wales Branch of the British Medical Association in our agitation for increased remuneration for medical officers employed by the New South Wales Government.

8. At the suggestion of certain influential private practitioners our Secretary was a candidate for election to the Council of the New South Wales Branch of the British Medical Association in March, 1937, but was unsuccessful. After a decent interval the committee renewed its request of former years for the direct representation of public medical officers on the Branch Council, as in England. On this occasion the proposal has been favourably received, and a decision is awaited.

9. After much consideration and consultation with groups and with the New South Wales Branch of the British Medical Association, a scale of salaries was drawn up for medical officers of the New South Wales Government, and was submitted to the Public Service Board by our Secretary at a preliminary investigation by the Board's senior inspector on October 5, 1937. On November 22 the Board heard our representatives, Dr. Latham, Dr. Nowland, and Dr. Willis, in support of the claim. On this occasion Sir Charles Blackburn and Dr. George Bell gave evidence on our behalf and stressed the necessity for improved conditions to attract the best type of graduate to the services. The Board reserved its decision.

10. During the year the staff of the Repatriation Department was brought by Act of Parliament under the Federal superannuation scheme. The terms are not as favourable as were desired, and have had a mixed reception by our repatriation group.

11. During the year the New South Wales Government established a new "Department of Social Services", and in it several medical officers have been appointed under unsatisfactory conditions. This matter is receiving attention by the committee in collaboration with the New South Wales Branch of the British Medical Association.

12. The association's delegates have again asked the Public Service Board for equal pay for male and female medical officers where both sexes are equally able to perform the duties of the position.

13. As indicated in our last report, the association's delegates early in the year had a long interview with the Director-General of Health of New South Wales, when several intra-departmental grievances were discussed and suggestions made for improving the status and prestige of medical officers in that department.

14. The difficulties experienced by medical officers of institutions in obtaining the small amount of leave allowed them by Regulation 152 have again received attention, and suitable representations have been made to the authorities concerned.

15. The committee has considered on several occasions the desirability and nature of an efficiency bar to promotion in the services, and has concluded that advancement beyond a salary of about £850 *per annum* should be contingent on the possession of some diploma or other appropriate post-graduate qualification.

Financial Statement.

Dr. C. E. Percy presented his financial statement for the year. It showed receipt of £23 5s. 6d. from subscriptions, and £8 15s. 1d. as interest on money on deposit, and expenditure of £7 17s. 3d. The amount in credit at the end of the year was £472 11s. 7d., being more than £5 per ordinary member.

Election of Office-Bearers.

The election of officers for the year 1938 resulted as follows:

President: Dr. A. A. Palmer.
Honorary Auditor: Dr. J. McGeorge.
Honorary Secretary: Dr. H. Hastings Willis.
Honorary Treasurer: Dr. C. E. Percy.

The following were elected by their respective groups as the committee for the year: Dr. C. E. Percy, Dr. J. Cooper Booth and Dr. J. McManamey (New South Wales Health Department), Dr. Edelston-Pope and Dr. W. K. Flook (Education Department), Dr. H. H. Nowland and Dr. S. Evan Jones (Mental Hospitals), Dr. G. L. C. Saunders (Works and Railways), and Dr. H. H. Willis (Repatriation Department).

Alteration of Rules.

Dr. H. H. Willis moved and Dr. C. E. Percy seconded the following motion for alteration of the rules, of which notice had been duly given:

> That there be added to Rule VII, paragraph III, as amended in 1935, the following sentence: "Any honorary member elected as an officer of the Association, as Auditor, or as a group representative on the Committee shall have the same duties, powers, responsibilities and privileges as if he were an ordinary member."

The motion was carried unanimously and the rules were declared altered accordingly.

Correspondence.

THE STATE AND THE MEDICAL PROFESSION IN QUEENSLAND.

SIR: The basis of the criticism of me which appeared in your editorial in THE MEDICAL JOURNAL OF AUSTRALIA of January 29 last appears to be threefold.

You complain:

1. That some of my arguments for the necessity of a revision of the relationships between the profession and the public are quoted (with minor alterations to suit Queensland's particular conditions) from the published statements of Sigerist, Sydenstricker, Parran and other accepted authorities; and that, moreover, you are in agreement with those authorities;

2. That the Queensland Branch of the British Medical Association has already proposed in general terms a policy for all Queensland, and that, as I know of this proposal, it should, irrespective of my official responsibilities, have been unchallenged;

3. That any "open letter" should, in defiance of its name, be "closed" and should be given no publicity.

The real basis of your editorial, however, seems to have been anxiety as to what personal, political or strategic significance (if any) might lie behind my letter.

As to the first charge, it is obvious to anyone who studies medical sociology that the admirable brevity of a sociologist, a medical historian and an administrative medical man, of international repute (like those quoted), modified in non-essentials to suit Queensland's conditions, is preferable to any paraphrase. The passages quoted are accepted generalizations and were quoted entire accordingly.

The quotation marks of the original were removed and the headings and leaded paragraphs were inserted by *The Telegraph* newspaper in the belief that the message of the letter would thus be more adequately conveyed. This can be confirmed by reference to Mr. A. Cummins, of that paper, who was the officer who handled the matter and with whose handling of it, in full knowledge of its object, I entirely agree.

Your slighting reference to "Doctor" Sydenstricker and to my comment on his being "perhaps the most gifted of the investigators" does no harm—to the late Edgar Sydenstricker, at any rate.

I. S. Falk, in his "Security against Sickness" (1936), rightly feels it an honour to dedicate that very up-to-date volume to the memory of a great man who did signal service both to the public and to the profession.

What a curious inversion of fact, too, it seems that you should feel that the term "Doctor" should not be applied to any Ph.D., LL.D., D.Sc. *et cetera*, but that the only connotation it could have is "Bachelor of Medicine"!

I am not surprised that you agree with the general content of the quotations—I used them because they were a matter of universal observation—but your sincerity would have been more apparent to those who know the authors if your own quotations had not stopped short so emphatically wherever they were about to refer to the conclusion of those investigators: that it is the public and the governments they elect, irrespective of medical men *as such*, that will decide finally how medical service is to be bought and paid for by that public and government.

Similarly, where you add that Sydenstricker says that several of his suggested solutions must be combined, you infer that this is omitted from my letter. Not only is it included, but the search for the means of such a coordination is the very basis of my appeal.

Moreover, the letter itself is a condensation of the paper on "The State, the Public and the Profession", which I was invited in 1936 by the Federal Health Council to deliver to the practising medical men of Hobart at the inaugural meeting of the National Health and Medical Research Council in 1937. You will recollect that the delegate to the council from the British Medical Association so vigorously opposed any private practitioner being invited to be present at that discussion of his own future that the paper was dropped. It had the sanction of the former President of the Queensland Branch of the British Medical Association, and in its present abridged form it has the endorsement in principle of the present Council of the British Medical Association here, which last month stated publicly that the letter was in general agreement with its own policy.

The charge that the Queensland Branch had (1935) adopted a general policy for all Queensland—a fact that I have the best of reasons for knowing—and that that fact should have paralysed my heretical pen simply shows (as the whole editorial does) a complete ignorance of the details of the Queensland situation. It is true that Queensland proposed a scheme of health insurance and had the utmost difficulty in forcing it through the Federal Council of the British Medical Association, but that scheme says nothing as to cost. A rough estimate in connexion with the complete scheme seems to show that it would involve an increase in the Unemployment Relief Tax in this State to some 4s. 6d. in the pound. Those who know Queensland will be aware that this burden would not fall upon the labourer, except perhaps indirectly, but would undoubtedly represent a new and possibly a major factor in unemployment.

Medical men must realize that a government may be wedded to a system of hospital provision as firmly as the British Medical Association has for two years been wedded ("for better or worse", as you added) to the scheme of medical insurance it opposed for twenty-five years after Lloyd George introduced it in 1911.

The service I regard as ideal is set out in my open letter. I have yet to learn that it differs in any way from the ideal expressed by the British Medical Association, but I have added to academic optima certain practical considerations that must inevitably modify the reality of the local proposals.

It was in the hope of receiving suggestions as to the best means of coordinating the divergent aspects of existing policies in Queensland that I appealed to the individual medical men—country practitioners as well as city specialists—and they responded most helpfully.

If, Mr. Editor, your labours leave you any time for reading, I suggest you read the "P E P" report so warmly applauded in *The British Medical Journal* in its editorial and digest of December 18, 1937. You will see that in England precisely the same viewpoint as to the necessity of a workable compromise between the hospital and the insurance policies is emphasized and enthusiastically welcomed.

Such a compromise cannot be reached here without honest cooperation with the Commonwealth and State Governments, and incompletely informed criticism of attempts in that direction is not only futile and disruptive, but may gravely prejudice the whole future of the medical profession.

I would recall to you those quotations with which you say you are in agreement, and refer you particularly to the comment of Parran that the future position of the profession will inevitably conform to the governmental framework.

As to the third charge that the "open letter" should have been "closed", and that in the form in which it was put out it invited publicity, has it occurred to you that an equal publicity was invited to the fact that 96% of the medical men expressed a ready adherence to any just and equitable system of cooperation? If you cannot guess the answer, therefore, to your question, I can only say that it would be a waste of time to attempt further enlightenment.

Why not work on the assumption that everyone is sincerely seeking a solution to one of the most difficult economic problems of the day—not here alone, but in Europe and America as well—and realize that suspicion and misrepresentation will only serve to breed their like. And next time you are in doubt, why not telephone!

Yours, etc.,

R. W. CILENTO.

Brisbane,
February 3, 1938.

THE CONTROL OF NARCOTIC DRUGS IN NEW SOUTH WALES.

SIR: Your article, "The Control of Narcotic Drugs in New South Wales", journal number 6, February 5, 1938, should impress members of the medical profession that regulations governing poisons should be observed.

It is true that the profession is deluged with drugs of all kinds; some of these are specialties which undoubtedly constitute an advance in medical therapeutics, others are indifferent, and some poor. In view of this, every physician should spare a few minutes from bridge or golf and make himself conversant with work done by eminent research workers in other countries. The cry that they have not time to do so is, of course, absurd.

With regard to samples sent to practitioners, mostly by request, for the past twelve months, immediately following the dispatch of a clinical package of barbituric acid, a special letter has been forwarded to each and every one of these medicos requesting an acknowledgement of safe receipt. Up to date, no communication or acknowledgement has ever been received from any member of the medical profession practising in the State of New South Wales.

Yours, etc.,
"$CH_3(CH_2)_{20}COOH$."

February 10, 1938.

Obituary.

JOHN BROOKE MOORE.

DR. JOHN BROOKE MOORE, whose death has been recorded in these pages, was widely known and respected in western New South Wales. He was an Irishman with a forceful and attractive personality, fearless in speech and ready in action. He was a cultured man with a delightful sense of humour, and in that he spared not himself in the service of his fellow man he was beloved by many.

Brooke Moore, the son of the late Mark Moore, medical practitioner of Cavan, Ireland, was born in that town in 1866. He studied medicine in his native land and in 1887 gained the licentiate of the King and Queen's College of Physicians of Ireland and of the Royal College of Surgeons of Ireland. Shortly after this he came to Australia, where he was appointed house surgeon to the Bathurst District Hospital. For a short period he practised at Wallsend and in College Street, Sydney, but for health reasons he returned to Bathurst, where he remained in practice till his death. When war broke out in 1914 Brooke Moore offered his services and did valuable work with the Australian Imperial Force. In 1928 he became a Fellow of the Royal Australasian College of Surgeons. He was, as might be expected, identified with many activities and organizations in and around Bathurst. His reputation as a surgeon was high and his personal qualities and human understanding enhanced that reputation. Steps are being taken in Bathurst to perpetuate his memory.

Dr. C. B. Howse writes:

It is now thirty years since I joined my brother, the late Neville Howse, in practice at Orange, and a few days after I arrived my brother said that he wanted me to drive down to Bathurst with him to meet his greatest friend in Australia, the late Brooke Moore. They had known each other for some years in the north of the State and then later in the west.

I have been in close touch with Brooke Moore since that first meeting thirty years ago, and he had an extremely fine personality, was liked by everyone, was the soul of hospitality, and, if it were necessary, would have given everything he possessed to help any of his friends in trouble.

He was extremely keen on his work, was a very good operator, and one of the pioneers of operative work in New South Wales. He did not care what he said, but never said an unkind word about anyone, or criticized their ability, irrespective of who they might be, whether his friends or otherwise, in opposition to him or otherwise. He was worshipped by his patients, whom he treated all the same, irrespective of their position, and the thing that interested him least of all was the financial part of his practice.

He was prouder of going to the Great War than anything else he had done in his life; he just left his practice and home, was not able to make any arrangements for the carrying on of his practice, but just went.

He had a long illness, which he faced as one would expect of him; just said that he was too old to go on living, and died without a complaint of any sort. To many hundreds of people Bathurst will not be the same with Brooke Moore gone; to those hundreds it is no longer Bathurst. I am glad to say that a committee has been formed to arrange about a memorial to Brooke Moore, the secretary being Mr. N. L. Bell, manager of the Bank of New South Wales, Bathurst.

PETER NEWTON MACGREGOR.

WE regret to announce the death of Dr. Peter Newton Macgregor, which occurred on February 9, 1938, at Sydney, New South Wales.

STANLEY CONNEBEE JAMIESON.

WE regret to announce the death of Dr. Stanley Connebee Jamieson, which occurred on February 10, 1938, at Kew, Victoria.

THOMAS JOHN MOORE KENNEDY.

WE regret to announce the death of Dr. Thomas John Moore Kennedy, which occurred on February 11, 1938, at Geelong, Victoria.

JOHN BERNARD GUNSON.

WE regret to announce the death of Dr. John Bernard Gunson, which occurred on January 25, 1938, at Adelaide, South Australia. (Unfortunately this name was reported in the issue of February 5 as that of Dr. James Bernard Gunson.)

PHILIP SYDNEY JONES.

WE regret to announce the death of Dr. Philip Sydney Jones, which occurred on February 17, 1938, at Sydney, New South Wales.

JAMES LOUIS EDGEWORTH SOMERS.

WE regret to announce the death of Dr. James Louis Edgeworth Somers, which occurred on February 17, 1938, at Mornington, Victoria.

Books Received.

LEAGUE OF NATIONS PUBLICATIONS. BULLETIN OF THE HEALTH ORGANISATION, Volume VI, Number 4; 1937. Geneva: Publications Department of the League of Nations; Australia: H. A. Goddard. Medium 8vo, pp. 176. Price: 2s. 6d. net.

INDIVIDUAL PSYCHOLOGY MEDICAL PAMPHLETS. NUMBER 18: THE MANAGEMENT OF EARLY INFANCY, PUBERTY AND ADOLESCENCE, THE PSYCHOLOGICAL APPROACH, AND THE NEUROTIC CHARACTER, by E. J. Partridge, H. Crichton-Miller, T. A. Ross and F. G. Crookshank; 1937. London: The C. W. Daniel Company Limited. Demy 8vo, pp. 60. Price: 2s. 6d. net.

ANOMALIES AND CURIOSITIES OF MEDICINE, by G. M. Gould, A.M., M.D., and W. L. Pyle, A.M., M.D.; 1937. New York: Sydenham, Publishers; Australia: Angus and Robertson Limited. Royal 8vo, pp. 968, with 295 illustrations in the text. Price: 30s. net.

FOOD AND PHYSICAL FITNESS, by E. W. H. Cruickshank, M.D., D.Sc., M.R.C.P., F.R.S.E., with a foreword by J. B. Orr, D.S.O., M.D., D.Sc., F.R.S.; 1938. Edinburgh: E. and S. Livingstone. Crown 8vo, pp. 160. Price: 5s. net.

LEAGUE OF NATIONS PUBLICATIONS. BULLETIN OF THE HEALTH ORGANIZATION. Volume VI, Number 5, October, 1937. Geneva: League of Nations Publications Department; Australia: H. A. Goddard. Medium 8vo, pp. 211. Price: 2s. 6d. net.

NEURO-OPHTHALMOLOGY, by R. L. Rea, B.Sc., M.D., M.Ch., F.R.C.S.; 1938. London: William Heinemann (Medical Books) Limited. Crown 4to, pp. 590, with numerous illustrations and coloured plates. Price: 42s. net.

AN INTRODUCTION TO PHYSICAL ANTHROPOLOGY, by E. P. Stibbe, F.R.C.S., with an appendix by W. A. M. Smart, M.B., B.S., B.Sc.; Second Edition; 1938. London: Edward Arnold and Company. Demy 8vo, pp. 237, with illustrations. Price: 10s. 6d. net.

Diary for the Month.

MAR. 1.—New South Wales Branch, B.M.A.: Organization and Science Committee.
MAR. 2.—Western Australian Branch, B.M.A.: Council.
MAR. 2.—Victorian Branch, B.M.A.: Branch.
MAR. 3.—South Australian Branch, B.M.A.: Council.
MAR. 4.—Queensland Branch, B.M.A.: Branch.
MAR. 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
MAR. 11.—Queensland Branch, B.M.A.: Council.
MAR. 15.—New South Wales Branch, B.M.A.: Ethics Committee and Medical Politics Committee.
MAR. 16.—Western Australian Branch, B.M.A.: Branch.
MAR. 22.—New South Wales Branch, B.M.A.: Council (Quarterly).
MAR. 23.—Victorian Branch, B.M.A.: Council.
MAR. 24.—South Australian Branch, B.M.A.: Branch.
MAR. 24.—New South Wales Branch, B.M.A.: Annual Meeting.
MAR. 25.—Queensland Branch, B.M.A.: Council.
MAR. 29.—New South Wales Branch, B.M.A.: Council (Election of Officers and Standing Committees).

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xvi to xviii.

ALFRED HOSPITAL, MELBOURNE, VICTORIA: Honorary Assistant Surgeon.

AUSTIN HOSPITAL FOR CANCER AND CHRONIC DISEASES, HEIDELBERG, VICTORIA: Honorary Physician.

DEPARTMENT OF MENTAL HYGIENE, MELBOURNE, VICTORIA: Medical Officer.

DOWERIN HOSPITAL BOARD, DOWERIN, WESTERN AUSTRALIA: Medical Officer.

ROYAL MELBOURNE HOSPITAL, VICTORIA: Assistant to Radiotherapist.

SAINT VINCENT'S HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Officers.

SYDNEY HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Officers.

THE UNIVERSITY OF MELBOURNE, VICTORIA: Chair of Anatomy.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

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